A PRACTICAL TREATISE ON

NERVOUS DISEASES .

PLATE I.

Right foot of T. S. after being pendent twenty minutes. (J. M. Taylor.)
A case of erythromelalgia. (See page 143.) By courtesy of Dr. S. Weir Mitchell.

A PRACTICAL TREATISE ON

NERVOUS DISEASES

FOR THE MEDICAL STUDENT AND GENERAL PRACTITIONER

BY

F. SAVARY PEARCE, M.D.

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE MEDICO-CHIRURGICAL COLLEGE
OF PHILADELPHIA; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA;
NEUROLOGIST TO THE PHILADELPHIA AND HOWARD HOSPITALS; MEM-
BER OF THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION,
AND OF THE AMERICAN CLIMATOLOGICAL ASSOCIATION;
CHAIRMAN OF SECTION ON NERVOUS AND MEN-
TAL DISEASES OF THE AMERICAN
MEDICAL ASSOCIATION

COLOURED FRONTISPIECE
NINETY-ONE ILLUSTRATIONS IN THE TEXT, MANY
IN COLOURS

NEW YORK AND LONDON
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DEDICATED TO

S. WEIR MITCHELL, M.D., LL.D.

WHOSE LIFE HAS BEEN AN INSPIRATION FROM CHILDHOOD

AND WHOSE FRIENDSHIP HAS BEEN OF

VALUE SINCE IN MEDICINE

PREFACE

THIS text-book on Nervous Diseases has been written for the student and general practitioner. It has ever been the object to curtail details of the doubtful points in neurology and to make most in small compass of practical facts in the study of nervous disorders. We believe that this is the surest method of instilling knowledge of so intricate a subject. The chapter on anatomy and physiology has been blended, so that its study will insure a pretty clear understanding of the anatomy and functions of the various parts of the nervous system. I have to thank my colleague, Prof. Isaac Ott, for the physiological details given. I beg to acknowledge, among many other authorities, special reference to the works of Gowers, Mills, Dana, Barker, Osler, and Strümpell, to the latter three of which the author is also indebted for several cuts, for which credit is given in the text. I gratefully acknowledge the kindness for the use of illustrations from The Diagnostics of Internal Medicine, by Dr. G. R. Butler, and Accident and Injury, by Dr. Pearce Bailey, respectively; also to Dr. M. K. Kassabian and Dr. G. E. Pfahler for the X-ray productions and some of the photographs.

It is the purpose of the author to follow this contribution by a similar treatise on Mental Diseases, so that the two works can be used in consonance by the medical student and busy practitioner.

F. S. P.

1409 LOCUST STREET, PHILADELPHIA, PA.

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A PRACTICAL TREATISE ON NERVOUS DISEASES

CHAPTER I

GENERAL ANATOMY, PHYSIOLOGY, AND CHEMISTRY OF THE NERVOUS SYSTEM

SECTION I.—ANATOMY

THE *structure* of the nervous system may be distinctly divided into five parts, as follows:

1. Non-medullated nerve-fibres.
2. Medullated nerve-fibres.
3. Neuroglia.
4. Supporting connective-tissue framework, besides the neuroglia.
5. Nerve-cells.

Classification of Nerves.—Nerve-fibres may be classified into three main groups, according to the direction in which they convey normally nervous impulses. These three divisions are:

1. Efferent nerve-fibres.
2. Afferent nerve-fibres.
3. Intercentral nerve-fibres.

Efferent or *centrifugal nerves* are those which conduct nervous impulses from the central nervous system (brain and spinal cord) to other parts of the body.

Afferent or *centripetal nerves* are those which conduct nervous impulses in the opposite direction—namely, from all parts of the body to the central nervous system.

Intercentral nerves are those nerves which connect nerve-centres together; they connect different parts of the brain and of the cord to one another.

Nerve-Centres.—Nerve-centres are composed of white and gray matter. In the brain the gray matter is on the outside, and in

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the spinal cord it is on the inside. The white matter is made up of nerve-fibres, and the gray matter of cells and an interposing cement substance called *neuroglia*. It is expedient to here explain what is meant by the *neuroglia*. This is the cementing tissue of the nervous system, of ectodermic origin—differing from connective tissue, which is solely derived from the mesoderm—and in the spinal cord it arises in part from the pia mater and passes into the white matter, carrying with it blood-vessels, and forming sæpta which separate the nerve-fibres into bundles. Neuroglia proper is made up of a delicate reticulum, holding in its meshes the small *glia cells*.

Nerve-Cells.—Nerve-cells differ greatly in size and shape. The nucleus is generally large and spherical, containing a distinct nucleolus. They may be roughly divided into three groups, according to the number of protoplastic processes they possess, into *unipolar, bipolar,* and *multipolar cells.*

Unipolar cells are found in the spinal ganglia. They are spherical in shape, are inclosed in a nucleated sheath, and the single process after a short course joins one of the nerve-fibres traversing the ganglion by a T-shaped junction.

Bipolar cells are cells with two branches or processes. The embryonic condition of the cells of a spinal ganglion is one example of these.

Multipolar Cells.—Here the cell becomes angular or stellate. It was formerly thought, in some instances, as in the cells of the sympathetic ganglia, that all the processes become nerve-fibres, but this is not so, for here, as well as in the large cells of the gray matter of the spinal cord, only one process becomes the axis cylinder of a nerve-fibre, the others dividing and subdividing in a ramified manner until they end in an arborescence of fine twigs.

The nerve-cell is the fundamental characteristic unit of the gray matter, and is known as a *neuron* (Waldeyer, 1891). Some of these cells are coloured or pigmented, such as in the *locus niger,* where they are black; *nucleus ruber,* where they are red, as its name would imply, etc. Every nerve-cell has an axis-cylinder process, which has been variously called the *axis cylinder, neurit, axone,* or *neuraxon.* The protoplasmic prolongations of the nerve-cells are called *dendrons.* According to the number of these processes, the nerve-cells are termed, as before stated, unipolar, bipolar, and multipolar.

DENDRITES — { Carry impulses to the cell
{ Degenerate if separated from it

CELL BODY — { Is the container and generator of
{ nerve force
{ Is trophic for dendrites and axones

AXONE
(axis cylinder) — { Carries impulses from the cell body
{ Degenerates if separated from it

COLLATERALS —

A = UPPER OR CEREBRAL MOTOR
NEURONE

EXERCISES AN INHIBITORY CON-
TROL OVER B

END BRUSH
(or terminal
arborization) — { Intermingles but does not unite with
{ dendrites of neurone to which it
{ carries impulses

B = LOWER OR PERIPHERAL MO-
TOR NEURONE

EXERCISES TROPHIC INFLUENCE
OVER AND MAINTAINS TONUS
OF MUSCLES

END ORGAN
in muscular fibre

FIG. 1.—The neurone (diagrammatic). (From Butler's Diagnostics of Internal Medi-
cine, 1902.)

The nerve-cells vary from $\frac{1}{4000}$ to $\frac{1}{250}$ of an inch in diameter. The largest nerve-cells are found in the anterior horns of the spinal cord and in the vicinity of the fissure of Rolando.

Nerve-Fibres.—The nervous system is composed of two parts—the *central nervous system* and the *peripheral nervous system*. The central nervous system consists of the brain and the spinal cord; the peripheral nervous system consists of the nerves which conduct the nervous impulses to and from the central nervous system, and thus bring the nerve-centres in relation with other parts of the body.

Nerve-fibres are of two histological kinds, viz., *medullated* and *non-medullated*. Medullated nerve-fibres are found in the white matter of the nerve-centres and in the nerves originating from the brain and spinal cord. Non-medullated nerve-fibres occur in the sympathetic system.

The *medullated* or *white fibres* are characterized by the sheath of white colour, fatty in nature, and stained black with osmic acid; it is called the *medullary sheath* or *white substance of Schwann*; this ensheaths the essential part of the fibre, which is a process from a nerve-cell, and is called the *axis cylinder*. According to Piersol, the axis cylinder is covered by a thin, transparent, elastic sheath, called the *axilemma*. The substance of Schwann is ensheathed by a thin, homogeneous membrane of an elastic nature, called the *neurilemma*.

The *axis cylinder* is made up of a number of small fibrils, which are held together by a cement substance called the *neuroplasm*.

Along the course of a medullated nerve-fibre there are numerous constrictions called the *annular constrictions* or *nodes of Ranvier*. At these constrictions the neurilemma lies in direct contact with the axis cylinder of said nerve. According to some authorities, the axilemma lies interposed. The stretch of nerve between two nodes is called an *internode,* and in the middle of each node is a *nucleus* which belongs to the primitive sheath. The nucleus is not necessarily always in the middle, for in some cases it is found to be variously distributed.

Incisures of Lantermann.—In each interannual segment in a nerve that is stretched there will be noticed a number of oblique lines running across the white substance of Schwann. This indicates that the segments of Schmidt are built up of a series of conical sections, each of which is bevelled at its ends, while the

slight interval between them appears as an incisure of Lanter-mann.

The fibres of the nerve-centres differ from the preceding in that they have no neurilemma and no annular constrictions. When a nerve enters the spinal cord or brain it loses its neurilemma.

The *non-medullated fibres* or *fibres of Remak* have no medul-lary sheath, and are therefore devoid of the double contour of the medullated fibres, and are unaffected in appearance by osmic acid. They consist of an axis cylinder covered by a nucleated fibrillated sheath. These nerves branch very frequently. They are princi-pally found in the sympathetic system, a few being found in the spinal nerves mixed with the medullated fibres.

Arrangement of Nerve-Fibres.—The arrangement of nerve-fibres is best seen in transverse section.

The nerve is composed of a number of bundles of *funiculi* of nerve-fibres bound together by connective tissue. The sheath of the whole nerve is called the *epineurium*; that of the funiculi the *perineurium*; that which passes between the fibres in a funiculus the *endoneurium*.

The *size* of a nerve-fibre is from $\frac{1}{5000}$ to $\frac{1}{1200}$ of an inch in diameter.

Nerve-Centres in General.—The nerve-centres in general are the *spinal cord, medulla oblongata, pons varolii, cerebellum,* and *cerebrum.*

Spinal Cord.—The spinal cord is enclosed in the bony canal formed by the spinal vertebræ; it extends from the margin of the foramen magnum to the lower border of the first lumbar vertebra, terminating in a slender filament of gray substance, the *filum terminale,* which lies in the midst of the roots of many nerves forming the *cauda equina.* It does not fill the canal altogether, but is suspended in the cerebro-spinal fluid. It has two enlarge-ments, one in the cervical, the other in the lumbar region. These are the situations whence the large nerves for the supply of the limbs issue. The spinal cord is from 15 to 18 inches in length and weighs about one ounce.

It is composed of white and gray matter; the white matter is situated externally, and constitutes its chief portion; the gray matter is in the interior, being so arranged that in a transverse section of the cord it appears like two crescentic masses which are connected together by a narrower portion which is known as
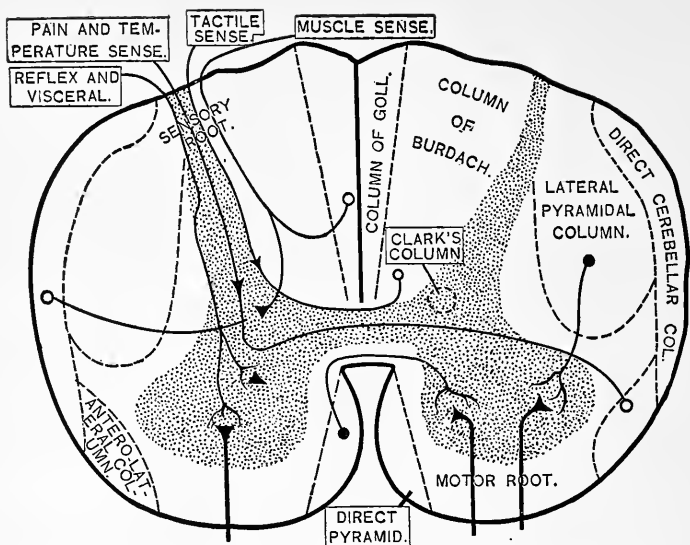
FIG. 2.—Showing the functions of the fibres of the anterior and posterior roots, and their relations to the horns and columns of the spinal cord. (Redrawn and modified from Dana.)
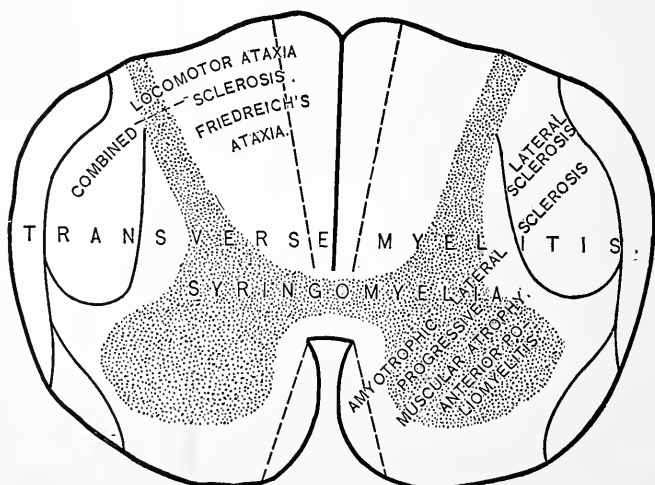
FIG. 3.—Showing the parts and columns of the spinal cord and some of the diseases which affect them. (From Butler.)

the *posterior commissure.* In the centre of this narrow portion is located the *central canal.* Directly in front of the posterior commissure is located the *anterior commissure,* which is composed of white matter.

The spinal cord consists of two lateral halves, separated anteriorly by the *anterior median fissure,* which extends from the front surface of the spinal cord to the anterior or white commissure. Posteriorly it is separated indistinctly by the *posterior median fissure,* which extends from the posterior surface of the spinal cord to the posterior or gray commissure. The posterior fissure is deeper than the anterior, but it is not as wide nor is it so distinct. The posterior fissure extends from the calamus scriptorius in the fourth ventricle above to the termination of the cord below.

On the lateral surface of the cord are noticed two longitudinal furrows, which divide each half of the cord into three columns, viz., *anterior, lateral,* and *posterior.* From the furrow between the anterior and lateral columns spring the anterior (*motor*) nerves; from the furrow between the lateral and posterior columns spring the posterior (*sensory*) nerves.

The cord is composed of a cortical white substance and a central gray matter. The white matter is made up of medullated nerve-fibres of different sizes, arranged longitudinally, and of a supporting material of two kinds, viz.: (*a*) Ordinary fibrous connective-tissue sæpta with elastic fibres, which is connected with sæpta from the pia mater, passing into the cord to carry blood-vessels; (*b*) neuroglia; the processes of the neuroglia cells are arranged so as to support the nerve-fibres which are without the usual external nerve-sheaths. The gray matter of the cord consists of nerve-fibres, most of which are very fine and delicate, of nerve-cells with branching processes, and of an extremely delicate network of the primitive fibrillæ of axis cylinders. This fine network is called *Gerlach's network,* and is mingled with the meshes of neuroglia. The neuroglia of the gray matter resembles that in white matter, but instead of everywhere forming a close network to support the nerve-fibres, here and there it is in the form of a more open sponge-work to support the nerve-cells. It is especially well developed around the central canal, being called here the *substantia gelatinosa centralis.* At the tip of the posterior horn it is also well developed, and is called *substantia gelatinosa lateralis of Rolando.* The latter is well developed in the cervical

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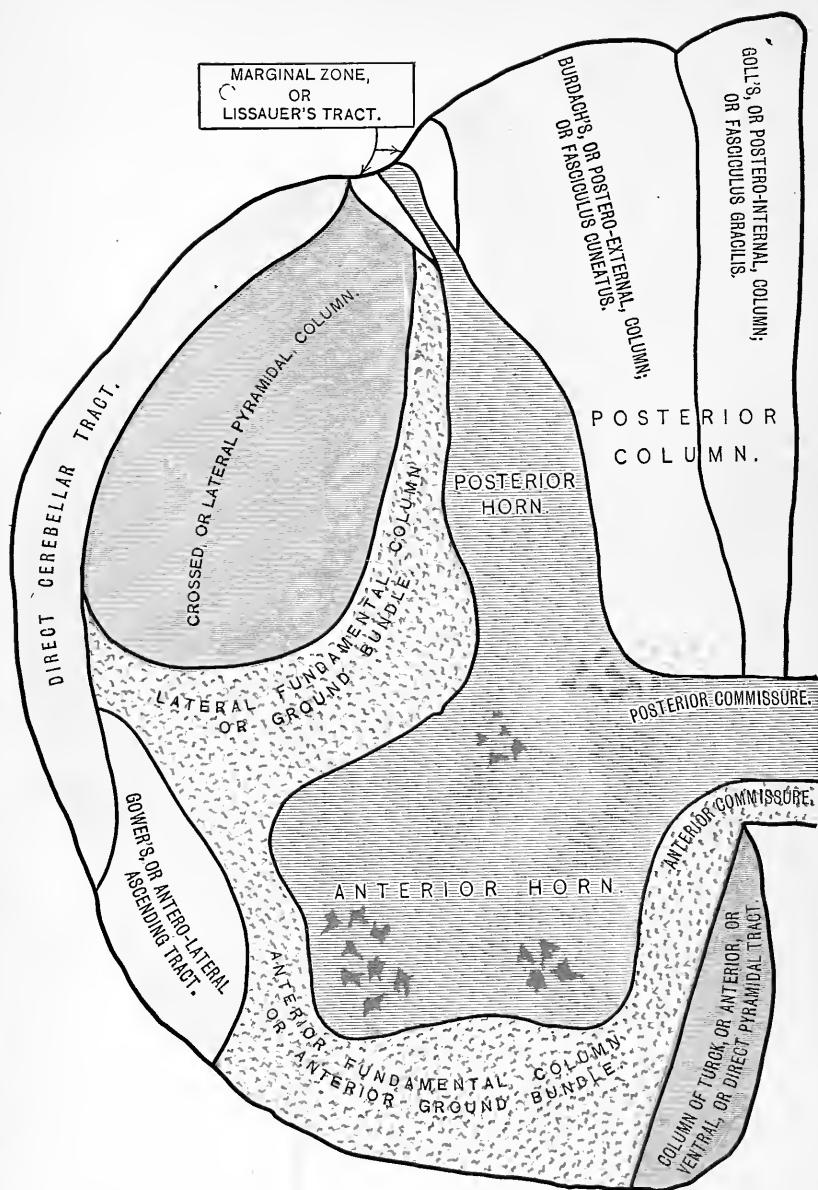
FIG. 4.—Showing the tracts of the spinal cord and their varied nomenclature.
Red = motor (or efferent). Blue = sensory (or afferent). (Butler.)

region. The gray substance is thicker in the lumbar and cervical regions than anywhere else. The gray substance of the horns or cornua causes the anterior horns to be thicker than the posterior. The anterior horns do not quite reach the free surface of the cord, while the posterior horns do.

The groups of cells in the gray matter are either scattered singly or arranged in groups, of which the following are to be distinguished on either side. The nerve-cells in the anterior horn of gray matter are principally large, multipolar, ganglion cells. They are arranged as a *mesial group,* near the inner aspect of the horn, and an *anterior* and a *lateral group.* The *column of Clarke* is a column of nerve-cells situated at the junction of the posterior horn with the gray commissure. These latter cells are seen only in the thoracic portion of the cord. The smallest nerve-cells are found in the posterior horns of the cord.

The fibres of the spinal cord are of two kinds—the *extrinsic* and *intrinsic.* The extrinsic fibres begin in the cerebrum of the brain, perhaps in the cerebellum, and run down the spinal cord to cells therein. The intrinsic fibres connect different parts of the spinal cord with each other.

Differentiation of Tracts.—The white matter can be systematically divided into different tracts by the following methods of observation:

1. *Embryological Method.*—It has been found by examining the spinal cord at different stages of its development that certain groups of fibres put on their myelin sheath at earlier periods than others, and that the different groups of fibres can therefore be traced in various directions. This is sometimes called the *Flechsig method.*

2. *Wallerian or Degeneration Method.*—This method depends upon the fact that if a nerve-fibre is separated from its nerve-cells it wastes or degenerates. It consists in tracing the course of tracts of degenerated fibres which result from an injury to any part of the central nervous system. When fibres degenerate below a lesion, the tract is said to be *descending degeneration,* and when the fibres degenerate in an opposite direction, the tract is one of *ascending degeneration.*

3. Another method is by removing an organ of special sense, and then tracking the degenerated fibres.
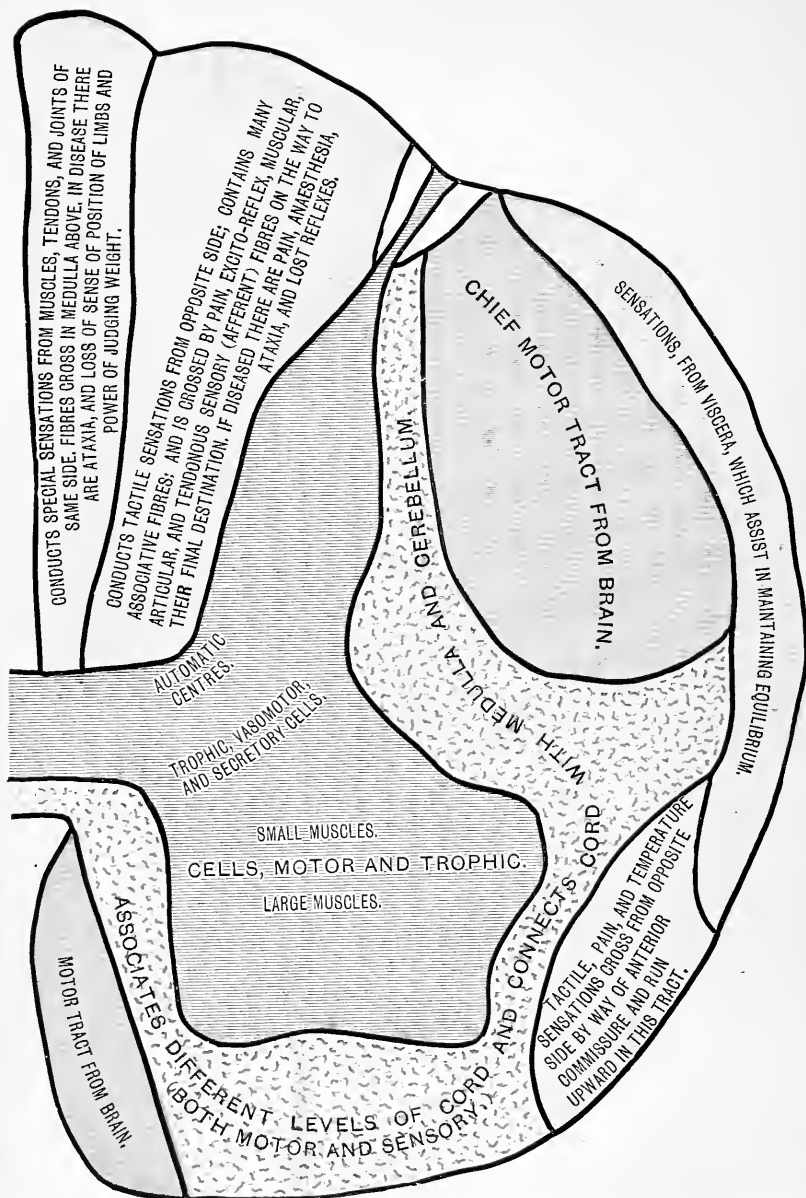
FIG. 5.—Showing the functions of the various tracts of the spinal cord. Red = motor (or efferent). Blue = sensory (or afferent). Compare with Fig. 4. (Butler.)

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Tracts discovered by the above Methods of Determination.— The spinal cord may be considered as a series of segments super-imposed one upon the other, corresponding to the pairs of spinal nerves. Each of these sections is a complete centre, is sup-plied with nerve-cells and with motor and sensory nerves. The nerve-cells are grouped into motor and sensory fields, united by the intraspinal fibres. These commissures or fibres are very short.

1. *Anterior Column.*—This is composed of fibres known as the commissural fibres, and the innermost contribute to the for-mation of the white commissure of the cord. There are two tracts located in the anterior column, viz.: on either side of the anterior median fissure a portion of the column is taken up by the *direct pyramidal tract* (fasciculus of Turk), which can be traced to be continuous with the non-decussating fibres of the pyramid of the medulla. The rest of the anterior column is composed of fibres of the *anterior lateral ground bundle.* The anterior column would be bounded in the following manner: internally by the an-terior median fissure, externally by the anterior horn and nerves springing from same, and anteriorly by the free surface of the cord. It may be stated that it is more or less triangular in shape. Some observers claim that the *anterior lateral ground bundle* is wholly situated in the lateral column.

2. *Lateral Column.*—This column is composed of fibres which are larger on the surface of the cord and smaller in the deeper portions. There is no decussation in the spinal cord of these fibres. It is made up of the following tracts, viz.: *crossed pyram-idal, direct cerebellar, anterior lateral ascending,* sometimes called tract of Gowers, *anterior lateral descending,* sometimes called tract of Löwenthal; *Lissauer, mixed lateral tract,* and the *an-téro-lateral ground bundle.* The direct cerebellar, or tract of Flechsig, is situated at the posterior superficial part of the lateral column. This extends downward as far as the second lumbar nerve, and upward as far as the restiform body, with which fibres it passes into the cerebellum. Gowers's tract occupies the anterior superficial surface of the lateral column. It commences in the lumbar swelling and terminates in the cerebellum, passing through the superior peduncles. The lateral column would be bounded in the following manner: internally by the anterior and posterior horns and nerves leading from the anterior horn; externally the

ROOTS	SPINOUS PROCESS	EXITS

C I   ARISES ABOVE ATLAS
  II   ARISES OPPOSITE ATLAS
  III   ARISES AT ATLAS
  IV   ARISES BETWEEN 2nd & 3rd SPINE
  V   "   OPPOSITE 3rd SPINE

BETWEEN ATLAS & OCCIPUT
ABOVE ATLAS

ABOVE 3rd CERVICAL SPINE

  VI   "   "   4th   "   "   4th   "   "
  VII   "   "   5th   "   "   5th   "   "
  VIII   "   "   6th   "   "   6th   "   "
D I   "   "   6th   "   "   7th   "   "
  II   "   "   7th   "
                                                  "   1st DORSAL SPINE
  III   "   "   1st DORSAL SPINE

  IV   "   "   2nd   "   "
BETWEEN 1st & 2nd DORSAL SPINE

  V   "   "   3rd   "   "   "   2nd " 3rd   "   "
  VI   "   "   4th   "   "   "   3rd " 4th   "   "
  VII   "   "   5th   "   "   "   4th " 5th   "   "
  VIII   "   "   6th   "   "   "   5th " 6th   "   "
  IX   "   "   7th   "   "   "   6th " 7th   "   "
  X   "   "   8th   "   "   "   7th " 8th   "   "
  XI   "   "   9th   "   "   "   8th " 9th   "   "
  XII   "   "   10th   "   "   "   9th " 10th   "   "
L I   "   10th " 11th   "   "
  II   }   "   "   11th   "   "
III   "   11th & 12th   "   "
  IV   }   "   "   12th   "   "   BETWEEN 11th
  V   DORSAL & 1st LUMBAR   "

                                  "   1st & 2nd   "   "

S I   "   2nd " 3rd   "   "
  II   }   "   "   1st LUMBAR   "
III   "   3rd " 4th   "   "
  IV   }
                                    "   4th " 5th   "   "
                                  BETWEEN 5th
                                LUMBAR & 1st SACRAL   "

Black Dots = Points of Origin.

Circles = Points of Exit.

C = Cervical.   D = Dorsal.

L = Lumbar.   S = Sacral.

  "   1st & 2nd   "   "
  "   2nd " 3rd   "   "
  "   3rd " 4th   "   "
  "   4th " 5th   "   "

FIG. 6.—Diagram showing the relation of the segments of the spinal cord, and of the roots and exits of the spinal nerves, to the spinous processes of the vertebræ.

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lateral column is comprised between the lines of implantation of the anterior and posterior roots.

3. *Posterior Column.*—This column is composed of fine fibres, and is remarkable for its abundance of neuroglia. It is made up of the following tracts—*Goll's* and *Burdach's.* The internal column, near the posterior fissure, is apparent at the upper part of the cord in the form of a slight enlargement. It is formed of long commissural fibres. The fibres rise as low down as the cauda equina, and run up to the nucleus of Goll in the medulla oblongata. The tract of Burdach is developed before the tract of Goll. Burdach's tract terminates in the nucleus of Burdach. The posterior column would be bounded in the following manner : internally by the posterior median fissure, externally by the posterior horn, and posteriorly by the free margin or surface of the spinal cord.

The *gray columns of cells* of the cord are *Clarke's,* and *intermedio-lateral* tracts.

Degenerations.—We now pass from this to consider the tracts of degeneration that occur when the spinal cord is cut right across in the thoracic region. Some tracts will be found degenerated in the piece of cord below the lesion ; these consist of nerve-fibres that are connected with the nerve-cells in the brain ; they are called the pyramidal tracts. Other tracts are found degenerated in the piece of cord above the lesion ; these consist of fibres that are connected with the nerve-cells of the spinal ganglia, or with the cells of the spinal cord itself below the lesion, and are passing upward.

The tracts which degenerate downward are the motor tracts ; the tracts that degenerate upward are the sensory tracts.

Tracts of Descending Degeneration.—1. *Crossed Pyramidal Tract.*—This tract is situated in the lateral column on the outer side of the posterior cornu of gray matter. At the lower part of the spinal cord it extends to the margin, but higher up it becomes displaced from this position by the interpolation of another tract of fibres, viz., the direct cerebellar tract. The crossed pyramidal tract is large, and may touch the gray matter at the tip of the posterior cornu, but is separated from it elsewhere. Its shape on cross-section is somewhat like a lens, but varies in different regions of the cord, and diminishes in size from the cervical region downward, its fibres passing off as they descend, to arborize around the nerve-cells and their branchings in the gray matter

of the cord. The fibres of which this tract is composed are moderately large, but are mixed with some that are smaller.

2. *Direct Pyramidal Tract.*—This tract is situated in the anterior column by the side of the anterior fissure. It is small in some animals, but is quite conspicuous in the human spinal cord. It can be traced upward to the brain, and downward as far as the mid or lower thoracic region, where it ends.

The two pyramidal tracts come down from the brain; in the medulla oblongata, the greater number of the pyramidal fibres cross over to the other side of the cord which they descend, hence the term "crossed pyramidal" tract; a smaller collection of the pyramidal fibres goes straight on on the same side of the cord, and these cross at different levels in the anterior commissure of the cord lower down; hence the disappearance of the direct pyramidal tract in the lower part of the cord.

3. *Anterior Lateral Descending Tract.*—An extensive tract, elongated but narrow, and reaching from the crossed to the direct pyramidal tract. It is a mixed tract, since not all of the fibres degenerate below the lesion.

4. *Comma Tract.*—This is a small tract of fibres which degenerate below section or injury of the cord. It is only found for a few millimetres below the actual lesion, though it degenerates downward; it is in reality a sensory tract, being composed of the branches of the entering posterior root-fibres which pass downward on entering the cord.

Tracts of Ascending Degeneration.—1. *Goll.*—This degenerates upward on injury or on section of the cord, as well as on section of the cord from below upward, and can be traced into the bulb. It is made up of very fine fibres.

2. *Direct Cerebellar Tract.*—This tract is situated on the outer part of the cord between the crossed pyramidal tract and the margin. It is found in the cervical, thoracic, and upper lumbar regions of the cord, and increases in size from below upward. It degenerates on injury or section of the cord itself, but not on section of the posterior nerve-roots. As its name implies, it passes up into the cerebellum. Its fibres are large, and originate from the cells of Clarke's column of the same side of the cord.

3. *Anterior Lateral Ascending Tract.*—This tract is situated at the margin of the cord outside the corresponding descending

tract.   Its fibres are of various sizes, and originate from cells situated in the base of the anterior horn of the opposite side of the spinal cord, and in the lower thoracic and lumbar regions; the fibres pass through the gray matter or commissure and anterior horn of the opposite side, and travels up as the tract of Gowers to terminate above, principally in the cerebellum.

4. *Lissauer's Tract.*—This is a small tract of ascending fibres, situated at the outer side of the tip of the posterior cornu.   It is made up of fibres of the posterior nerve-roots.

Complete section of the spinal cord (transverse) leads to:

(*a*) Loss of motion of the parts supplied by the nerves below the section on both sides of the body.   The paralysis is not confined to the voluntary muscles, but includes the muscular fibres of the blood-vessels and viscera.   Hence there is a fall of blood-pressure, paralysis of sphincters, etc.

(*b*) Loss of sensation in the same regions.

(*c*) Degeneration, descending and ascending, on both sides of the cord.

Hemisection.—If the operation performed is not a complete cutting of the spinal cord across transversely, but a cutting of half the cord across, it is termed hemisection.   This operation leads to:

(*a*) Loss of motion of the parts supplied by the nerves below the section on the same side of the body as the injury.

(*b*) Loss of sensation in the same region.   The loss of sensation is not a very important symptom, and is limited to the sense of localization and the muscular sense.   The animal can still feel sensations of pain and of heat and cold.

(*c*) Degeneration, ascending and descending, nearly entirely confined to the same side of the cord as the injury.

A lesion of the pyramidal tract in the cord may cause *hemiplegia* or *paralysis* of one arm or leg, called *monoplegia*.

When the motor decussation takes place in the anterior pyramids, it cuts off the anterior horns of the spinal cord, forming a number of nuclei which give origin to motor fibres of the cranial nerves.   The posterior horns are cut off by the decussation of the fillet and give sensory nuclei which form origins to the cranial nerves.

Medulla Oblongata.—The medulla oblongata is the upper expended part of the spinal cord.   It connects the upper part of the

spinal cord with the pons varolii. The weight of the medulla is about 100 grains. In the medulla, the fibres from the cord are rearranged, the gray matter is also much changed, while new gray matter is added. Each half of the medulla consists of the following parts, from before backward: *anterior pyramid, olivary body, restiform body, funiculus gracilis,* and *funiculus cuneatus.* By the divergence of the posterior columns and the restiform bodies, the floor of the fourth ventricle is formed. At the anterior lower border of the medulla we find the decussation of the pyramids, where the fibres cross over to the lateral columns of the cord. The *anterior pyramid* receives the direct pyramidal tract of the anterior column of the cord from its own side, and the crossed pyramidal tract from the lateral column of the cord of the opposite side. Most of the pyramidal fibres pass through the pons directly to the cerebrum, a few fibres passing to the cerebellum. The anterior median fissure of the spinal cord terminates at the foramen cæcum, just beneath the pons. In transverse section at the level of the inferior olive we see the pyramids in front. External to the pyramid, on either side, is the inferior olive with its contained nucleus of gray matter called the *dentate nucleus.* The *formatio reticularis* is seen in the centre of the section. *Formatio reticularis* is an association system of short fibres which is to be met with at any point between the spinal cord and the thalamus. These fibres run at right angles to each other. Imbedded in it are nerve-cells.

The *restiform body* is composed of fibres from the following sources :

From the superior olive.

From lateral nucleus.

From the direct cerebellar tract of the same side.

From the nucleus gracilis and nucleus cuneatus of the same side (posterior superficial arcuate fibres).

From the nucleus gracilis and nucleus cuneatus of the opposite side (anterior superficial arcuate fibres).

From lateral nucleus of opposite side.

From the olivary nucleus by fibres which pass through the opposite olive, superficial to the opposite olive, and deeper than the opposite olive.

The *funiculus gracilis* of the medulla is composed of fibres which occupy the *column of Goll* in the spinal cord.

The *funiculus cuneatus* is composed of fibres which occupy the *column of Burdach* in the spinal cord.

The *funiculus of Rolando* is the continuation upward into the medulla of the gelatinous substance capping the posterior horns in the spinal cord.

The *medulla* is about 1 inch in length, ¾ of an inch in width, and ½ an inch in thickness. It has four faces.

The triangular space between the restiform bodies is a part of the floor of the *fourth ventricle*. Running transversely across the floor of the fourth ventricle are a group of fibres which give partial origin to the auditory nerve. They form the " beard " of the fourth ventricle. Just at the divergence of columns of Goll is the nib of the fourth ventricle, called the *calamus scriptorius*.

Centres found in the Medulla.—Respiratory, cardio-inhibitory, diabetic, deglutition, vaso-motor, cardio-accelerator, vomiting, salivation, mastication.

Pons Varolii, Etc.—This is situated between the medulla and the crura cerebri. It is a white body in the form of a half ring. Its size depends upon the size of the hemisphere with which it has to do. It weighs about 250 grains. It has six faces. The anterior face rests upon the basilar process of the occipital bone, and presents a median groove for the basilar artery. The posterior surface of the pons forms a part of the floor of the fourth ventricle.

The pons is formed of scattered nerve-fibres and cells. The transverse fibres which form the cortex go for the most part through the cerebellar peduncles to the hemispheres.

There are three planes of transverse fibres: first, the superficial, which covers the pyramidal columns; second, the one known as the deep stratum, which separates the pyramidal tracts from the fillets; and the third, known as the *complex,* which separates the different tracts. On transverse section we see that the organ is divisible into an anterior portion or *crusta,* and a posterior portion or *tegmentum.* The *superior olive* is a collection of gray matter. The *trapezium* is a prominent group of transverse fibres running from the accessory nucleus and tuberculum acusticum to the superior olive situated in front of the tegmentum. Numerous small masses of gray matter are found in the pons, which are known as the *pontine nuclei.*

The pons is a cross-way for the conduction of motion in one direction and sensation in the opposite; and besides this function of the pons there is a co-ordination centre in the pons for reflex actions. Irritation of the pons will cause convulsions; therefore it contains *a convulsive centre*.

Cerebral Peduncles.—The cerebral peduncles extend from the superior part of the pons to the optic thalami, and their size is in direct relation with the size of the brain proper. They are about ¾ of an inch in length. After leaving the pons they are separated from each other, each going to the hemisphere of the respective sides. The space between them is known as the *interpeduncular space*. The cerebral peduncles have an anterior face, which presents a longitudinal groove. The external face is embraced in great part by the hippocampal convolution.

Upon transverse section we see a black-looking structure which separates the crusta from the tegmentum and is known as the *locus niger*, dividing the pes or crusta from the tegmentum. The tegmentum consists of masses of gray matter and fibres extending through the posterior end of the medulla oblongata, pons, and crura up to the optic thalami. The *crusta* is composed of two bundles, the internal or cortico-pontal, which goes to the anterior portion of the brain, and which might be considered as a commissure of the brain, connecting that portion with the gray nuclei in the medulla and pons and the cerebellum; and another, the external bundle or voluntary motion bundle, which comes from the ascending frontal and ascending parietal convolutions, and ends in the anterior horns of the spinal cord. The external bundle contains the geniculate bundle. This starts in the cortex around the speech centre, and ends in the hypoglossal, facial, and trigeminal nuclei.

In the peduncle is seen the *nucleus ruber,* which is composed of a mass of red fibres and cells, and in which ends the superior cerebellar peduncle.

In the tegmentum of the peduncle are also sensory fibres. In the tegmentum are three masses of longitudinal fibres, viz., the posterior, the central, and superior.

There are three fillets. The *median* or upper, which rises from Goll and Burdach's nuclei and runs on through the tegmentum chiefly, and in lateral nucleus of optic thalamus, and then enters the cortex of the brain. The *lateral fillet,* which starts from Goll and Burdach's nuclei, runs to the superior olive and

ends in the posterior corpus quadrigeminum. It is composed of fibres concerned in hearing. The *mesial fillet* comes from the same source as the others. It terminates in the base of cerebrum.

Capsules of the Brain.—They are the *internal* and *external*. The *internal capsule* is composed of a band of white fibres, being divided into an anterior and a posterior segment. The point where the segments are united is known as the *genu*. The *internal capsule* is bounded internally by the optic thalamus and caudate nucleus, externally by the lenticular nucleus. The fibres composing the anterior segment of the internal capsule are the cortico-pontal fibres anteriorly and the geniculate tract posteriorly. In other words, the geniculate tract lies just in front of the genu. The posterior segment of the internal capsule is made up of *motor* fibres for its anterior two-thirds, and *sensory* for the posterior one-third. The motor tract has the following origin and distribution: From ascending frontal, ascending parietal convolutions (motor area), through anterior two-thirds of posterior segment of internal capsule, thence through crura, pons, anterior pyramids of medulla, and there decussate. Crossed pyramidal portion goes down opposite lateral column; direct pyramidal portion goes down the same side, both tracts ending finally in the anterior horns for further distribution. The sensory tract is traced as follows: From occipital, parietal, and temporal lobes, through posterior third of internal capsule (posterior segment) into peduncle, divides, the main part goes through tegmentum to nuclei of Goll and Burdach, down posterior columns through ganglion of posterior root to skin. The rest or remainder of the sensory fibres go through the lateral columns for further distribution.

The *external capsule* is a band of fibres bounded laterally or internally by the lenticular nucleus and externally by the claustrum.

Cerebellum.—The cerebellum is composed of an elongated central portion or lobe, called the *vermiform process,* and two lateral *hemispheres.* Each hemisphere is connected with its fellow not only by means of the vermiform process, but also by a bundle of fibres called the *middle peduncle* (the latter forming the greater part of the transverse fibres of the pons), while the *superior peduncles,* which decussate in the mid-brain, connect it with the cerebrum, and the *inferior peduncles* (restiform bodies) connect it with the medulla oblongata.

The cerebellum is composed of white and gray matter, the latter being external, like that of the cerebrum, and, like it, infolded so that a larger area may be contained in a given space. The tree-like arrangement of the white matter has given rise to the name *arbor vitæ*. Besides the gray matter on the surface, there are in the centre of the white substance of each hemisphere small masses of gray matter, the largest of which is called *corpus dentatum*. The others are called *nucleus globosus, nucleus fastigii,* and *nucleus emboliformis*.

If a section is taken through the cortical portion of the cerebellum, the following distinct layers can be seen by the microscope.

Underneath the pia mater is the *external layer* of gray matter; it is formed chiefly of fine nerve-fibres with small nerve-cells scattered through it. Into its outer part processes of pia mater pass vertically; these convey blood-vessels. There are also here numerous long tapering neuroglia cells. The *internal* or *granular* layer of gray matter is made up of a large number of small nerve-cells mixed with a few larger ones, and some neuroglia cells. Between the two layers is an incomplete stratum of large flask-shaped cells called the cells of *Purkinje*. Each of these gives off from its base a fine process which becomes the axis cylinder of one of the medullated fibres of the white matter; the neck of the flask passing in the opposite direction breaks up into dendrites, which pass into the external layer of gray matter. Each cell of Purkinje is further invested by arborizations of two sets of nerve-fibres. One of these (originating from the fibres of the white matter which are not continuous as axis cylinders from the cells of Purkinje) forms a basket-work round the dendrons; the other (originating as axis-cylinder processes from the nerve-cells of the external layer) forms a felt-work of fibrils round the body of the cell.

The cells of the internal layer of gray matter are small; their dendrites intermingle with those of neighbouring cells; their axones penetrate into the external layer, but their final destination is uncertain. Ramifying among these cells are fibres characterized by possessing bunches of short branches at intervals.

Cerebrum.—The cerebrum consists of two halves called cerebral hemispheres, separated by a longitudinal fissure and connected by a large band of transverse commissural fibres known as

the *corpus callosum*. The interior of each hemisphere contains a cavity of complicated shape called the *lateral ventricle*.

Each hemisphere is covered with gray matter, which passes down into the fissures that abound on its exterior. The amount of this gray matter varies directly with the amount of convolutions of the surface. Under it white matter is situated, and at the base there are masses of gray matter; parts of these *basal ganglia* are seen forming part of the wall of the ventricles. The anterior basal ganglion is called the *corpus striatum*; it is divided into two parts, called the *lenticular nucleus* and the *caudate nucleus*. The posterior basal ganglion is called the *optic thalamus*.

Passing between the basal ganglia are the white fibres, which enter the cerebral hemispheres from the crus; these constitute the *internal capsule* (already explained).

The brain cortex has four layers, as follows: 1, molecular layer, containing caudal cells; 2, layer of small pyramidal cells; 3, layer of large pyramidal cells and cells of Martinotti whose axones run upward to the first or molecular layer; 4, layer of polymorphous cells.

The outer or external surface of the brain presents numerous depressions and elevations, the latter being known as *convolutions*. Each cerebral hemisphere is divided into lobes, which are further divided by many depressions or *fissures* into convolutions. The cause of folding of the cerebral substance is due to the rigidity of the cranium during development.

There are five great fissures, viz., the great longitudinal, the great transverse, fissure of Sylvius, fissure of Rolando, and the occipito-parietal.

The *fissure of Rolando* commences at the great longitudinal fissure, and runs downward, terminating above the horizontal limb of the fissure of Sylvius.

The *weight* of the brain is about 52 ounces. In rare cases it may reach 65 ounces. It is heavier in civilized than uncivilized races; greater in the male than in the female.

Optic Thalami.—The optic thalami are about the size of pigeons' eggs, and are directed obliquely forward and inward, and approach each other at their anterior extremities. Between them lies the *third ventricle*.

The thalami have three nuclei, viz., *external*, or lateral nucleus of Flechsig, *internal*, and *anterior*. The lateral nuclei are

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the points of attachment of the posterior roots. The other nuclei do not have anything to do with the posterior roots.

There are some cells between the *pulvinar* and the origin of the *peduncle of the pineal gland,* called the *habenula.* There is also a nucleus known as the nucleus of the habenula.

Corpora Quadrigemina, Etc.—These are four small bodies, separated from the *aqueduct of Sylvius* by the *lamina quadrigemina.* The anterior pair are connected by the superior brachia with the *external geniculate bodies.* The posterior pair are connected by the inferior brachia to the *internal geniculate bodies.* The anterior corpora quadrigemina are principally concerned in sight, while the posterior are concerned principally in hearing.

Corpus Striatum.—This is so called from the fact that white fibres of the internal capsule pass through it. They are situated a little in front and slightly outward from the optic thalami. The nuclei composing these bodies, as before stated, are the lenticular and caudate.

Membranes of the Brain and Cord.—The brain and spinal cord are enveloped in three membranes: 1, *dura mater*; 2, *arachnoid*; 3, *pia mater.*

1. The *dura mater* or external covering is a tough membrane composed of bundles of connective tissue which cross at various angles, and in whose interstices branched connective-tissue corpuscles lie; it is lined by a thin, elastic membrane on the inner surface of which is a layer of endothelial cells.

2. The *arachnoid* is a more delicate membrane, very simple, and similar in structure to the dura mater, and lined on its outer free surface by an endothelial membrane.

3. The *pia mater* consists of two chief layers, between which numerous blood-vessels ramify. Between the arachnoid and the pia mater is a network of fibrous tissue trabeculæ sheathed with endothelial cells; these *subarachnoid trabeculæ* divide up the *subarachnoid* space into a number of irregular sinuses. There are some similar trabeculæ, but fewer in number, traversing the subdural space, i. e., the space between the dura mater and arachnoid.

SECTION II.—PHYSIOLOGY

Physiology of the Nervous System.—*Functions of the Spinal Cord.*—It is a great conducting medium, carrying impulses up-

ward and downward, and within itself from side to side; it is the great *reflex centre,* or rather series of so-called reflex centres. Impulses originate within it a function of minor importance, however.

Spinal Reflexes.—By *reflex action or movement* is meant a movement caused by the stimulation of an afferent (sensory) nerve. The simplest definition is: a *reflex action* is an afferent impulse followed by an efferent impulse. Another definition is: a *reflex act* is the transmission of irritation by the neuraxone of a sensory neurone to the dendrones of the motor neurone, and by its neuraxone to the muscle.

The stimulus, on being applied to an *afferent nerve,* sets up a state of excitement (nervous impulse) in that nerve, which state of excitement is transmitted or conducted in a centripetal direction along the nerve to the centre (spinal cord); where the nerve-cells represent the *nerve-centre* in the cord, the impulse is transferred to the motor, *efferent,* or centrifugal channel. *Three* factors, therefore, are essential for a reflex motor act—*afferent fibre,* a *transferring centre,* and an *efferent fibre*; these together constitute a *reflex arc.*

In a purely reflex act all voluntary activity is excluded. Reflex movements may be divided into three classes, as follows:

a. The *simple* or *partial reflexes,* which are characterized by the fact that stimulation of a sensory area discharges movement in one muscle only, or at least in one limited group of muscles.

b. The *extensive inco-ordinate reflexes,* or *reflex spasms.* These movements occur in the form of clonic or tetanic contractions; individual groups of muscles, or all the muscles of the body may be implicated.

c. Extensive co-ordinated reflexes are due to stimulation of a sensory nerve, causing the discharge of complicated reflex movements in whole groups of different muscles, the movements being "*purposive*" in character, i. e., as if they were intended for a particular purpose.

If an electric irritant is applied to a motor nerve there will be a greater contraction of the muscle than if the irritant was applied directly to the muscle itself.

In reflex action are three elements: first, the external irritation; second, excitation of nerve-centres themselves; third, contractions of the muscles.

The *seat* of reflex action is mainly in the cord, although some centres are found in the medulla and pons varolii.

Pflüger's Laws of Reflex Action.—1. The reflex movement occurs on the same side on which the sensory nerve is stimulated, while only those muscles contract whose nerves arise from the same segment of the spinal cord.

2. If the reflex occurs on the other side, only the corresponding muscles contract.

3. If the contractions be unequal upon the two sides, then the most vigorous contractions always occur on the side which is stimulated.

4. If the reflex excitement extend to other motor nerves, those nerves are always affected which lie in the direction of the medulla oblongata. Lastly, all the muscles of the body may be thrown into contraction.

In the spinal cord *reflex impressions* pass at the rate of 20 *feet* per second.

Inhibition of the Reflexes.—Within the body there are mechanisms which can suppress or inhibit the discharges of reflexes, and they may therefore be termed mechanisms inhibiting the reflexes. These are:

1. *Voluntary Inhibition.*—Reflexes may be inhibited voluntarily, both in the region of the spinal cord and brain. Example: Keeping the eyelids open when the eyeball is touched; arrest of movement when skin is tickled. We must observe, however, that the suppression of the reflexes is possible only up to a certain degree.

2. *Setschenow's Inhibitory Centres.*—They are located in the corpora quadrigemina and the optic thalami.

3. *Strong stimulation of a sensory nerve* inhibits reflex movements. The reflex does not take place if an afferent nerve be stimulated very powerfully. Example: Suppressing a sneeze by friction of the nose; suppression of the movements produced by tickling, by biting the tongue.

4. *Poisons.*—Chloroform diminishes the reflex excitability by acting upon the centres.

A constant current of electricity passed longitudinally through the cord diminishes the reflexes, especially if the direction of the current is from above downward.

Some *drugs* affect the reflex excitability directly by acting on

the spinal cord—e. g., *methylconine*—but other drugs may produce the same result indirectly by affecting the heart and the blood supply to the cord. If the abdominal aorta be compressed for a few minutes to cut off the blood supply to the cord and lower limbs, *paraplegia* is temporarily produced. Blood is absolutely necessary for the maintenance of function in the cord. If the supply be cut off from the brain, the person will lose consciousness in four seconds. Anæmia will stimulate reflex actions for a certain time. If the excitation is carried to a gréat degree the cord will become fatigued. If the spinal cord is cut in the lumbar region there will be a rhythmic action of the sphincters. *Strychnine* is poisonous to every nerve-cell, while *chloroform* is poisonous to every cell both of plants and animals.

According to *Brown-Séquard*, reflex activity is most marked in birds, amphibians, reptiles, next in mammals and fishes. In the new-born it is always very great.

The physician, by studying the condition of the reflexes, can form an idea as to the condition of (practically) every inch of the spinal cord. The following reflexes have been noted:

1. *Plantar Reflex.*—Obtained by tickling the sole of the foot. Centre in the lumbar region.

2. *Patellar Reflex.*—Obtained by striking the tendon above or below the patella. Centre between second and third lumbar nerves.

3. *Cremasteric Reflex.*—Obtained by tickling or pinching the inside of the thigh; the testicle is drawn up. Centre in the lumbar region, between second and third lumbar nerves.

4. *Abdominal Reflex.*—Obtained from a sharp push in the abdomen, causing contraction of the abdominal muscles. Centre between eighth and twelfth dorsal nerves.

5. *Epigastric Reflex.*—If the skin is excited between the fourth, fifth, and sixth intercostal spaces, there will be a contraction of the rectus abdominis. Centre between fourth and eighth dorsal nerves.

6. *Scapular Reflex.*—On irritating the skin over the scapula there will be a contraction of the shoulder muscles. Centre between seventh and eighth cervical and second dorsal nerves.

7. *Cilio-spinal Reflex.*—If the skin of neck is pinched there will be a dilatation of the pupil. Centre between sixth cervical and third dorsal nerves.

Other Reflex Centres in the Spinal Cord.—*a. Ano-spinal* centre, or centre controlling the act of defecation.

b. Vesico-spinal centre for regulating micturition.

c. Erection or *genito-spinal* centre, located in the lumbar region. The afferent nerves are the sensory nerves of the penis; the efferent nerves for the deep artery of the penis are the vaso-dilator nerves arising from the first to the third sacral nerves.

d. Ejaculation Centre.—The afferent nerve is the dorsal nerve of the penis; the centre lies at the fourth lumbar vertebra; the motor fibres of the vas deferens arise from the fourth to the fifth lumbar nerves, which pass into the sympathetic, and from thence into the vas deferens. The motor fibres for the bulbo-cavernosus muscle, which ejects the semen from the bulb of the urethra, lie in the third and fourth sacral nerves.

e. Vaso-motor Centres.—Both vaso-constrictor and vaso-dilator centres are distributed throughout the whole of the spinal axis.

f. Sweat Centres.—These are located in the spinal cord.

g. Parturition Centre.—This lies at the first and second lumbar vertebra; the afferent fibres come from the uterine plexus, to which also the motor fibres proceed. Goltz observed that a bitch became pregnant after its spinal cord was divided at the first lumbar vertebra.

Excitability of the Spinal Cord.—Even at the present time observers are by no means agreed whether the spinal cord, like the peripheral nerves, is excitable, or whether it is distinguished by the remarkable peculiarity that most of its conducting paths and ganglia do not react to direct electrical and mechanical stimuli. It is contended by most observers that if stimuli be cautiously applied either to white or gray matter, there is neither movement nor sensation. As the spinal cord conducts to the brain impulses communicated to it from the stimulated posterior roots, but does not itself respond to stimuli which produce sensations, Schiff has applied to it the term *æsthesodic*. Further, as the cord can conduct both voluntary and reflex motor impulses without, however, itself being affected by stimuli applied to it directly, he called it *kinesodic*.

The following views have been expressed:

1. In the *posterior columns* the sensory root-fibres of the posterior root which traverse these columns give rise to painful impressions, but the proper paths of the posterior columns them-

selves do not do so. Removal of the posterior column produces *anæsthesia* (loss of tactile sensation). *Algesia* (or the sensation of pain) remains intact, although at first there may even be *hyperalgesia*.

The *trophic centres of posterior roots* are the posterior root ganglia.

2. The *anterior columns* are non-excitable, both for striped and non-striped muscle, as long as the stimuli are applied only to the proper paths of these columns. But movements may follow either when the anterior nerve-roots are stimulated or when, by the escape of the current, the posterior columns are affected, whereby reflex movements are produced.

The *trophic centres of anterior roots* are the anterior horns.

3. The *vaso-constrictor nerves,* which proceed from the vaso-motor centre and run downward in the lateral columns of the cord, are excitable by all stimuli along their whole course; direct stimulation of any transverse section of the cord constricts all the blood-vessels below the point of section. In the same way, the fibres which ascend in the cord and increase the action of the vaso-motor centre are also excitable. Stimulation of these fibres, although it affects the vaso-motor centre reflexly, does not cause sensation.

4. *Chemical stimuli,* such as the application of salt or wetting the cut surface with blood, appear to excite the spinal cord somewhat.

5. The *motor centres* are directly excited by blood heated to about 40° C., or by asphyxiated blood, or by sudden and complete anæmia of the cord produced by ligature of the aorta, and also by certain poisons. *Picrotoxin, nicotine,* and compounds of *barium* seem also to produce the effect.

Paths of Spinal Cord for Pain, Heat, and Cold; Muscular and Tactile Sensations.—*Pain, heat,* and *cold* impressions pass through the gray matter of the spinal cord from cell to cell.

Muscular and *tactile sensations* are transmitted through or by the white matter of the cord.

Vaso-motor nerves come down the lateral columns in the gray substance to the anterior roots.

The nerves from the *centre of respiration* run through the lateral columns and then enter the gray substance of the anterior horns and leave by the anterior roots.

The *sweat and inhibitory fibres* running down the lateral columns both decussate, the former in the cord and the others in the medulla. The sweat fibres pass out from the anterior roots, also the branches which make up the splanchnics.

There is one *sensory decussation* in the fillet fibres.

There are two *motor decussations*: the lower one is in the cord, in the anterior commissure, while the higher is in the anterior pyramids.

Locomotor Ataxia.—In locomotor ataxia there is a degeneration of the lower part of the posterior column. Tactile and muscular sensations are abolished. Pain, heat, and cold sensations, however, are still transmitted, as a rule.

Syringomyelia.—This is a disease of the gray tract, with gliosis and final cavity formation. Sensations of heat, pain, and cold are abolished, while tactile and muscular sensations are still transmitted.

When there is a lesion in the pons, there is a paralysis of the face on one side and of the arm and leg on the opposite side. This is known as *crossed hemiplegia.*

Monoplegia is paralysis of certain areas alone, as in the leg, arm, etc., due to hæmorrhage in the brain or disease of certain parts of the motor area.

There is a plentiful supply of blood-vessels in all portions of the brain. Both the caudate and lenticular nuclei are supplied by the *middle cerebral artery.* There are three lenticular branches; one of these is known as the *lenticulo-striate artery of cerebral hæmorrhage.* It is sometimes called *Charcot's artery* of cerebral hæmorrhage.

The brain has no true lymphatics, but both in the brain and cord are the perivascular lymph spaces of His which carry the lymph.

Nerve-Fibres, Function of.—A nerve wave is a transmission of nerve force through the axis cylinder of a nerve. This brings out the properties of nervous excitability. The laws of *nervous conductivity* are: First, the integrity of the nerve; the neuraxone must be intact. Second, isolated conduction; a nerve-fibre carries an impression without its being diffused to other nerves. Third, the law of conduction in both directions; if a nerve is irritated, the impression may be carried both ways.

Sensory and motor nerves are of the same nature and structure, but they are attached to different forms of apparatus.

Electricity travels ten million times faster than nerve waves.

Irritability is a fundamental property of nerves. After a nerve is cut and separated from the circulation, it will retain its irritability for some time, but eventually loses it. The Esmarch bandage will cause this phenomenon. When a nerve is cut, the first thing is a rise of irritability, soon followed by its loss. Woorara paralyzes the terminations of the motor nerves in the muscles. In symmetrical gangrene there is a slow stoppage of the circulation, and the pain is very acute. The patient suffers first from hyperæsthesia, but this soon disappears. When a mixed nerve is irritated, there is first an anæsthesia and then a paralysis. Heat causes nervous excitability and cold renders a part insensible, and if carried to excess causes temporary paralysis. Inflammation causes great irritation to the nerves. When such nerves are incised there is much more pain than in healthy tissue. Nerves possess the property of being thrown into a state of excitement by *stimuli,* and are therefore said to be excitable. The stimuli may be applied to and may act upon any part of the nerve. The following are the various kinds of stimuli—i. e., modes of motion—which act upon the nerves:

1. *Mechanical stimuli* act upon nerves when they are applied with sufficient rapidity to produce a change in the form of the nerve particles, such as pressure, pinching, tension, puncture, etc. In the case of *sensory* nerves, when they are stimulated pain is produced, as is felt when a limb " sleeps," or when pressure is exerted upon the ulnar nerve at the bend of the elbow. When a *motor* nerve is stimulated, motion results in the muscle attached to the nerve. If the continuity of the nerve-fibres be destroyed, the conduction of the impulse across the injured part is interrupted. *Continued pressure* upon a mixed nerve paralyzes the motor fibre sooner than the sensory fibres.

2. *Thermal Stimuli.*—If a frog's nerve be heated to 45° C., its excitability is first increased and then diminished. The higher the temperature, the greater is the excitability and the shorter is its duration. If a nerve be heated to 50° C. for a short time, its excitability is abolished as well as its conductivity. Sudden cooling of a nerve to 5° C. acts as a stimulus, causing contraction in a muscle.

3. *Chemical Stimuli.*—These excite nerves when they act with a certain rapidity and thereby alter the condition of the nerve.

Most chemical stimuli act by first increasing the nervous excita-
bility and then diminishing or paralyzing it. Chemical stimuli, as
a rule, have less effect upon the sensory than upon the motor fibres.
The following chemical stimuli excite nerves: alkaline salts, sugar,
urea, glycerin, and some metallic salts. Atropine diminishes the
action of the pneumogastrics and the glandular nerves, etc. Pilo-
carpine has the opposite action. Free alkalies, mineral acids
(not phosphoric), acetic, oxalic, tartaric, and lactic acids dimin-
ish the excitability of nerves, as well as most salts of the heavy
metals.

4. *Physiological* or *normal stimuli* excite the nerves in the
normal body. Its nature is entirely unknown. The "nerve
motion" thereby set up travels either in a "centrifugal" or out-
going direction from the central nervous system, giving rise to
motion, inhibition of motion, or secretion, or in a "centripetal"
or ingoing motion or direction from the specific end organs of the
nerves of the special senses or the sensory nerves.

5. *Electrical Stimuli.*—The following forms of electrical stim-
uli may be used: *a. A constant current,* which may be made or
broken. *b. Induction shocks,* either make or break shocks. *c. An
interrupted current.*

Constant Current.—If the constant current be used as a nerve
stimulus, the stimulating effect on the *sensory nerves* is most
marked at the moment of making and breaking the current; dur-
ing the time the current passes only slight excitement is perceived,
but even under these circumstances very strong currents may
cause very considerable, and even unbearable, sensations. If a
constant current be applied to a *motor nerve,* the greatest effect
is produced when the current is made or closed and when it is
broken or opened. But while the current is passing, the stimu-
lation does not cease completely; for, with a certain strength of
stimulus the muscle remains in a state of tetanus.

An irritation of sensory nerves, except *nervous depressor*, pro-
duces increase of blood pressure.

Induction Current or Shock.—If a galvanic element is closed
by means of a short arc of wire, at the moment the circuit is again
opened or broken a slight spark is noticed. If, however, the cir-
cuit is made or closed by means of a very long wire rolled in a
coil, then, on breaking the circuit, there is a strong spark. If the
wires be connected to two electrodes, so that a person can hold

one in each hand, the current at the moment it is opened must pass through the person's body—then there is a violent shock communicated to the hand. This phenomenon is due to a current induced in the long spiral of wire which has been called the *extra current.*

Interrupted or Faradic Current.—If a very long insulated wire be coiled into the form of a spiral roll, which is called the *secondary spiral,* and if a similar spiral, the *primary spiral,* be placed near the former, and the ends of the wire of the primary spiral be connected with the poles of a constant battery, every time the current in the primary circuit is made (closed), or broken (opened), a current takes place, or, as it is said, is induced, in the secondary spiral. If the primary circuit be kept closed, and if the secondary spiral be brought nearer to or removed farther from the spiral (primary), a current is also induced in the secondary spiral. The current in the secondary circuit is called the *Faradic current.* When the primary circuit is closed, or when the two spirals are brought nearer each other, the current in the secondary spiral has a direction opposite to that in the primary spiral, while the current produced by opening the primary circuit or by removing the spirals farther apart has the same direction as the primary. During the time the primary circuit is closed, or when both spirals remain at the same distance from each other, there is no current in the secondary spiral.

When a *galvanic current* is passed through a nerve, it is said to be in a state of *electrotonus,* because its irritability is modified. In this condition, the vital properties of the nerve are modified— i.e., its *electro-motivity,* its *excitability.*

If a nerve be so arranged upon the electrodes that its transverse section lies on one and its longitudinal on the other electrode, then the galvanometer indicates a strong current. If, now, a constant current be transmitted through the end of the nerve projecting beyond the electrodes, and if the direction of the current coincides with that in the nerve, then the magnetic needle gives a greater deflection, indicating an increase of the nerve current—*the positive phase of electrotonus.* The increase is greater the longer the stretch of nerve traversed by the current, the stronger the galvanic current, and the less the distance between the part of the nerve traversed by the constant current and that on the electrodes.

If in the same length of nerve the constant current passes in the opposite direction to the nerve current, there is a diminution of the electro-motive force of the latter—*negative phase of electrotonus.*

If two points of the nerve equidistant from the equator be placed on the electrodes, there is no deflection of the galvanometer needle. If a constant current be passed through one free projecting end of the nerve, then the galvanometer indicates an electro-motive effect in the same direction as the current (constant).

These experiments show that a constant current causes a change of the electro-motive force of the part of the nerve indirectly traversed by the constant current—i. e., in the intrapolar area—and also in the part of the nerve outside the electrodes—i. e., in the extrapolar area. This condition is called *electrotonus.*

Muscle Current during Electrotonus.—The constant current also produces an electrotonic condition in the muscle; a constant current in the same direction increases the muscle current, while one in an opposite direction weakens it, but the action is relatively feeble.

Cause of Electrotonus.—If a certain stretch of a living nerve be traversed by a constant electrical current, it passes into a condition of altered excitability which is known as *electrotonus.* This condition of altered excitability extends not only over the part actually traversed by the current, but it is communicated to the entire nerve. At the *positive pole* or *anode* the excitability is diminished; this is the region of *anelectrotonus.* At the *negative pole* or *cathode* the excitability is increased; this is the region of *cathelectrotonus.* The changes of excitability are most marked in the regions of the poles themselves.

Transmission of Nervous Impulses.—If a *motor nerve* be stimulated at its central end (1) a condition of excitation is set up, and (2) an *impulse* is transmitted along the nerve to the muscle with a certain velocity. The latter depends upon the former and represents the function of *conductivity.* According to Helmholtz the velocity for a human motor nerve-wave is about 100 feet per second.

In the *sensory nerves* of man, the velocity of the impulse is probably about the same as in motor nerves. The rates usually given are about 150 feet per second.

Relay Stations of Motor and Sensory Fibres.—The *optic thalami* are the relay stations of the *sensory tract*.

The *caudate nuclei* are the relay stations for the *motor tract*.

Chemical and Mechanical Properties of Nervous Substance.— Specific gravity of white substance, 1.040; gray substance, 1.050. The reaction is sometimes slightly alkaline and sometimes slightly acid. It contains a varying amount of water and solid substances; 75 per cent water and 25 per cent solid matter is about the average.

Proteids.—Albumin occurs chiefly in the axis cylinder and in the substance of the ganglionic cells. Some of this proteid substance represents characters not unlike those of myosin. Dilute solution of common salt extracts a proteid from nervous matter, which is precipitated by the addition of much water and also by a concentrated solution of common salt. *Potash albumin* and a *globulin-like* substance are also present. *Nuclein* occurs especially in the gray matter, while *neuro-keratin,* a body containing much sulphur and closely related to keratin, occurs in the neuroglia.

Fats and other allied substances soluble in ether, more especially in the white matter:

a. Cerebrin, free from phosphorus. It is a white powder composed of spherical soluble granules (in hot alcohol and ether), but insoluble in cold water. When boiled for a long time with acids, it splits up into a left rotatory body like sugar and another unknown product.

b. Lecithin and its decomposition products—glycero-phosphoric acid and oleo-phosphoric acid.

c. Protagon, which contains nitrogen and phosphorus, is similar to cerebrin and lecethin combined.

d. Leucin, inosite, etc.

The *physical properties* of nerve tissue are *cohesion* and *elasticity.*

Physiology of the Cerebellum.—Irritation of the cerebellum will cause no contractions or pain, but if it be deeply injured there will be a tottering of the animal called a *cerebellar tottering.*

If the cerebellum is entirely removed the animal will live for quite a time, and will soon learn to co-ordinate its movements. The cerebellum is the *centre of co-ordination* of muscular movements. It is probable that the co-ordinating fibres go through the descending antero-lateral tract.

Irritation of the cerebellum will not only cause a tottering walk, but also may cause a tendency to vomit, on account of the irritation of the vomiting centre in the medulla. Injury of the middle cerebellar peduncle causes a tendency for the animal to go to one side (*circus movements*). It also brings on internal squint on the same side and superior squint on the opposite side.

Lesions of one hemisphere may not give rise to any symptoms; but if the middle lobe is involved, there is inco-ordination of movements, especially a tendency to fall, unsteady gait, and pronounced vertigo. Irritative lesions of the middle peduncle cause complete gyrating movements of the body around its axis, together with rotation of the eyes and head.

Functions of the Medulla Oblongata.—The medulla oblongata, which connects the spinal cord with the brain, has many points of resemblance with the former. Like the cord, it is concerned in the *conduction* of impulses. In it numerous reflex centres are present—e. g., for simple reflexes similar to the nerve-centres in the spinal cord. There are other centres present which seem to dominate or control similar centres placed in the cord—e. g., the great vaso-motor centre, pupil-dilating centres, and the centre for combining the reflex movements of the body. Some of the centres are capable of being excited reflexly. It is also said to contain *automatic centres.* The normal functions of the centres depend upon the exchanges of blood and gases effected by the circulation of the blood through the medulla. If this gaseous exchange be interrupted or interfered with—as asphyxia, sudden anæmia, or venous congestion—these centres are first excited, and exhibit a condition of increased excitability, and at last, if they are over-stimulated they are paralyzed. An excessive temperature also acts as a stimulus by an action on the polypnœic centre in the tuber cinereum which drives the respiration centre into activity. All the centres, however, are not active at the same time, and they do not all exhibit the same degree of excitability. Normally, the respiratory centre and the vaso-motor centre are continually in a state of activity.

. The centres found in the medulla have been given under the anatomy of same.

The *respiratory centre* lies in the medulla behind the superficial origin of the vagus, on both sides of the posterior aspect of the apex of the calamus scriptorius, between the nuclei of the

vagus and accessorius. It consists of two parts, which are in a state of activity alternately, an *inspiratory* and an *expiratory,* each one forming the motor central point for the acts of inspiration and expiration. Some observers claim that this is an automatic centre, for after section of all the sensory nerves which can act reflexly upon the centre, it still retains its activity. The degree of excitability and the stimulation of the centre depend upon the state of the blood, and chiefly upon the amount of blood gases.

The Cardio-inhibitory Centre.—The fibres of the vagus, when moderately stimulated, diminish the action of the heart; when strongly stimulated, however, they arrest its action and cause it to stand still in diastole; they are supplied to the vagus through the spinal accessory nerve, and have their centre in the medulla oblongata. Gaskell has shown that stimulation of the vagus not only influences the rhythm of the heart's action, but modifies the other functions of the cardiac muscle. *Stimulation of the vagus* influences the automatic rhythm—i. e., the rate at which the heart contracts automatically; the force of the contractions, more especially the auricles, although in some animals—e. g., the tortoise— the ventricles are not affected; the power of conduction—i. e., the capacity for conducting the muscular contractions. This centre may be excited directly in the medulla, and also reflexly by stimulating certain efferent nerves. *Stimulation of the trunk of the vagus* from the centre downward, along its whole course, and also of certain of its cardiac branches, causes the heart either to beat more slowly or arrests its action in diastole. The result depends upon the strength of the stimulus employed; feeble stimuli slow the action of the heart, while strong stimuli arrest it in diastole.

Cardiac-Accelerating Centre.—This centre is also located in the medulla oblongata, which sends accelerating fibres to the heart. They pass from the medulla through the spinal cord, and leave the cord through the rami communicantes of the lower cervical and upper six dorsal nerves to pass into the sympathetic system. If the vagi of an animal be divided, stimulation of the medulla oblongata, of the lower end of the divided cervical spinal cord, even of the lower cervical ganglion, or of the upper dorsal ganglion of the sympathetic, causes acceleration of the heart-beats in the dog and rabbit without the blood-pressure undergoing any change. The inhibitory fibres of the vagus lose their excitability

more readily than the accelerating fibres, but the vagus fibres are more excitable than those of the accelerans.

Vaso-motor Centre and Nerves.—The chief dominating centre, which supplies all the non-striped muscles of the arterial system with motor nerves, lies in the medulla oblongata at a point which contains ganglionic cells. The nerves passing from this centre are known as the vaso-motor nerves, there being two kinds of them—*vaso-constrictor* and *vaso-dilator*. The vaso-constrictor nerves, as their name implies, constrict the arteries, or rather reduce their mean diameters, thus causing an increase of blood-pressure, resulting in the swelling of veins and the heart. The vaso-dilator nerves, as their name implies, cause a dilatation of the arteries and a decrease in blood-pressure.

Functions of the Pons Varolii.—This is seated in front of the medulla, and is the centre of *convulsive movements*. In epilepsy there is a lesion of the cortex of the brain, and the impressions are conveyed to the convulsive centre. It is also a path for the conduction of sensibility and motion. If there is a lesion in the pons, there is a paralysis of the face on one side and of the leg and arm of the opposite side. This is due to a lesion *below* the facial decussation and *above* the motor decussation of the fibres in the pons. The convulsion centre can be excited by excess of carbonic acid, lack of oxygen, anæmia, etc.

Functions of Cerebral Peduncles.—They are about ¾ of an inch in length, and contain fibres which connect the brain with the structures at its base. In the *locus niger* there is located the *high detrusor centre* of the bladder, although the proper *detrusor centre* is located down in the cord.

Injury to one cerebral peduncle causes, in the first place, violent pain and spasm of the opposite side, while the blood-vessels on that side contract and the salivary glands secrete. These phenomena of irritation are followed by paralytic symptoms of the opposite side—viz., anæsthesia and paresis. In affections of the cerebral peduncle in man, we must remember the relation of the oculomotorius to it, as the latter is often paralyzed on the same side, while the extremities, tongue, and half the face are paralyzed on the opposite side from the lesion.

Functions of the Corpora Quadrigemina.—Destruction of these bodies on one side in mammals causes actual blindness, which may be on the same or the opposite side, according to the relation

of the fibres crossing at the optic chiasm. Total destruction causes blindness of both eyes. At the same time, the reflex contraction of the pupil, due to the stimulation of the retina with light, no longer takes place where the optic is the afferent and the oculo-motorius the efferent nerve. If the cerebral peduncles are left in position and the cerebral hemisphere alone be removed, the pupil still contracts to light, as well as after mechanical stimulation of the optic nerve. Destruction of the corpora quadrigemina inter-feres with the complete harmony of the motor acts; disturbance of equilibrium and inco-ordination of movements occur.

In man very little is known regarding the effects of disease of the corpora quadrigemina, interference with the ocular mus-cles being the most marked symptom; but the inco-ordination of movement which has been observed may be due to pressure upon the superior cerebellar peduncle, while it is by no means certain that the defects of vision are directly due to lesions of these bodies.

Forced Movements.—It is evident from what has been said regarding the importance of the corpora quadrigemina for the harmonious execution of movements, that unilateral injury of such parts as are connected with them by conducting channels must give rise to peculiar unilateral disturbance of the equilibrium, causing variations from the symmetrical movements of both sides of the body. These are called " forced movements."

Strabismus and Nystagmus.—Among the forced movements may be reckoned deviation of the eyeballs, strabismus or squinting, and involuntary oscillation of the eyeballs, constituting *nystagmus.* The latter condition occurs after superficial lesions of the resti-form bodies, as well as of the floor of the fourth ventricle.

·*Contraction centre of iris* is in the anterior corpora quadri-gemina.

Functions of the Corpora Striata.—*Lenticular and Caudate Nu-clei.*—Electrical stimulation of these nuclei causes general con-tractions in the opposite half of the body which are due to simul-taneous stimulation of the neighbouring cortico-muscular paths. There is no sign of pain. Lesions of the lenticular nucleus or of the caudate nucleus do not seem to give rise to any permanent symptoms, provided the internal capsule is not injured. If the corpus striatum is destroyed on one side, there is paralysis of the opposite side of the body. This body holds the same relation to

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the motor tract that the optic thalami bear to the sensory tracts;
on the other hand, or in other words, the corpora striata are the
relay stations of the *motor tracts.*   These bodies also contain a
thermogenic centre, and if section is made of both the temperature
will rise to 110.5° F.   If section be made of the *tuber cinereum*
there will be a great increase of temperature.

Functions of Optic Thalami.—Ferrier did not observe any
movements on stimulating the optic thalami with electricity.   As
the *pulvinar* or the posterior extremity of the optic thalamus is in
part the origin of the optic nerve, and is also connected by fibres
with the cerebral cortex, it is probably related to the sense of
sight.   Injury to its posterior third in man results in disturbance
of vision.   Ferrier surmises that the sensory fibres pass through
the optic thalami on their way to the cortex, so that when they
are destroyed insensibility of the opposite half of the body is
produced.   Thus these bodies are called *relay stations* for the
sensory tracts.

The Capsules: Functions, Etc.—In connection with the func-
tions of the basal ganglia it is most important to remember their
relation to the internal capsule.   The *anterior segment* of the
internal capsule sweeps between the caudate and lenticular nuclei,
while the *posterior segment* lies between the optic thalamus and the
lenticular nucleus.   External to the first division of the lenticular
nucleus is the *external capsule,* whose function is unknown.   Ex-
ternal to this latter structure is the *claustrum,* whose function
also is unknown.   It is evident that hæmorrhage into or about the
basal ganglia is apt to involve the fibres of the internal capsule.
When the " artery of hæmorrhage " ruptures it may not only
destroy the lenticular nucleus, but the internal capsule will be
compressed, and the same is the case with the *lenticulo-thalamic
artery*—the external capsule will tend to force the blood inward.
We know that in the posterior segment of the capsule the voli-
tional or pyramidal fibres lie in the following order from before
backward : those for the face (and tongue) in the knee; in the
anterior third, those for the arm and hand; and in the middle
third, for the leg, and perhaps behind these those for the trunk;
so that a very small lesion in this region will affect a large num-
ber of these fibres, converging as they do like the rays of a fan
from the motor cortical areas, where the arrangement of these
centres is a supero-inferior one, to become an antero-posterior one

in the knee and posterior limb of the internal capsule. The posterior third of this limb is sensory, and is the *sensory cross-way*.

Destruction of the internal capsule causes paralysis of motion or sensibility, or both, on the opposite side of the body, according to the part of it which is injured.

Functions of Cerebral Hemispheres.—The cortical *motor areas* for the face, arm, and leg are grouped around the fissure of Rolando, including the ascending frontal, ascending parietal, and paracentral lobules. The centre for the face occupies the lowest third of the ascending frontal convolution, and reaches also to the lowest fifth of the ascending parietal. The arm centre occupies the middle third of the ascending frontal and middle three-fifths of the ascending parietal convolutions, while the leg centres lie at the upper end of the sulcus and extends backward into the parietal lobule. The leg centre is continued over on to the paracentral lobule, opposite the upper end of the fissure of Rolando, in the marginal convolution on the mesial aspect of the hemisphere, where the centres for the muscles of the trunk also exist. The motor areas are supplied by branches of the Sylvian artery.

To locate the fissure of Rolando on the cranium, a line is drawn from the glabella to the external occipital protuberance. Divide the distance into two equal parts, and $\frac{1}{2}$ an inch behind the middle point is the top of the fissure of Rolando.

The *cortical sensory areas* are distributed very irregularly. There must be some connection between the surface of the brain and the afferent channels through which sensory impulses pass inward; and although the channels for these impulses are, perhaps, not so well known, it must be that sensory impulses for the opposite half of the body travel upward through the posterior third of the posterior limb of the internal capsule, to radiate in all probability into the occipital, temporal, and parietal lobes. Parts of these different convolutions are sometimes spoken of as *sensory centres*.

Extirpation and *stimulation* are the methods employed to determine the different areas on the brain.

Hemiplegia consists of motor paralysis of one-half of the body, although, as a rule, all the muscles are not paralyzed to the same extent; in some there may be complete paralysis—i. e., they are entirely removed from voluntary control—while in others there is merely impaired voluntary control. It may be caused by affections of the cortical areas or by lesion of the motor tracts above

the medulla; and the paralysis is always on the side opposite to the lesion, owing to the decussation of the motor paths in the medulla. If the case be a severe one, there is a *complete hemiplegia* due to lesion of the cortical centres for the face, arm, and leg. While the arm and leg are completely paralyzed, the lower part of the face is more affected than the upper half, which is usually not affected. The arm recovers more quickly than the leg.

Conjugate deviation of the eyes, with rotation of the head, is frequently present in the early period of hemiplegia, although it usually disappears. When a person turns his head to one side there is an associated movement of certain of the ocular muscles with those of the neck. The head and eyes are usually turned to the side of lesion; this is termed *conjugate deviation,* so that the power of voluntarily moving the eyes and head to the paralyzed side is temporarily lost. The unopposed muscles rotate the head and eyes to the sound side. If a lesion be in the posterior part of the pons, the deviation is to the paralyzed side.

Irritation of the Motor Centres.—If the motor centres are irritated by pathological processes, such as hyperæmia, inflammation in a syphilitic diathesis, tumours, tubercles, cysts, fragments of bone, etc., there arise spasmodic movements in the corresponding muscle groups. This condition of a sudden discharge of the gray matter resulting in local spasms is called *Jacksonian cerebral epilepsy.*

The Centre of Speech.—The investigations of Broca and others have shown that the *third left frontal convolution* of the cerebrum (*Broca's area*) is of essential importance for speech, while probably the island of Reil is also concerned.

Three activities are required for speech: (1) the normal movement of the vocal apparatus; (2) a knowledge of the signs for objects and ideas; (3) the correct union of both.

Aphasia.—Injury of the speech centre causes either a loss or more or less considerable disturbance of the power of speech. The loss of the power of speech is called *aphasia.* Aphasia, as usually understood, means the partial or complete loss of the power of articulate speech from cerebral causes. A person may comprehend what he sees or hears, yet he is unable to speak.

Ataxic Aphasia.—Here there is loss of speech owing to inability to execute the various movements of the mouth necessary for speech and co-ordinated grimaces. He can utter inarticulate words or

sounds. The muscles concerned in articulation are not paralyzed, but there is an absence of co-ordination of these muscles due to disease of the cortical centre. Hence the patient cannot repeat what is said to him. Nevertheless, the psychical processes necessary for speech are completely retained, and all words are remembered; and hence these persons can still give expression to their thoughts graphically by writing. If, however, the finely adjusted movements necessary for writing are lost, owing to an affection of the centre for the hand, then there arises at the same time the condition of *agraphia,* or inability to execute those movements necessary for writing. Such a person, when he desires to express his ideas in writing, only succeeds in making a few unintelligible scrawls on the paper.

In *word blindness,* the person cannot name a letter or a word, so that he cannot understand symbols, such as printed or written words, or it may be a familiar object; although he can see quite well, while he can speak fluently and write correctly.

In *word deafness*, the person hears other sounds and is not deaf, but he does not hear and understand words. (See APHASIA.)

An animal or person can live with only one *cerebral hemisphere* intact; but if both are removed, the animal lies still, remaining in one position, and will not move.

Seats or centres of consciousness and volition (will) are located in the *cerebral cortex.*

The cerebrum contains the centres of intelligence. There are many ways of measuring the grade of intelligence of a man; the important is the *facial angle measurement,* explained as follows: The *facial angle* (Camper) is the divergence between a line drawn from the inferior anterior nasal ridge of upper jaw to the glabella, and another drawn from the nasal ridge to the external auditory meatus. In the Caucasian race it is 80°; in the Mongolian, 75°; in the Ethiopian, 70°; and in apes, 40°.

The Visual Centre.—According to Munk, this includes the occipital lobes (*cuneus*), while, according to Ferrier, it also includes the angular gyrus. In cases of long-standing blindness there has been noticed to be a consecutive disappearance of the occipital convolutions on both sides of the parieto-occipital fissure. Stimulation of the centre gives rise to the phenomena of light and colour. Injury causes a disturbance of vision, especially hemiopia of the same side, to be further explained under the eye.

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When one centre is the seat of irritation, there is a photopsia of the same halves of both eyes. Stimulation of both centres causes the occurrence of the phenomena of light or colour or visual hallucinations in the entire field of vision.

The *auditory centres* are found in the superior temporal convolutions. When this is completely removed, deafness is the result, while partial injury causes psychical deafness. It has been found that softening of the first temporal convolution is the cause of these phenomena.

The *centres of taste and smell* are located in the gyrus uncinatus.

The *centre of tactile sensibility* is located in the gyrus fornicatus, according to some observers; by others, in the motor area.

Sleep and Waking.—In sleep and waking we observe the periodicity of the active and passive conditions of the brain. During sleep there is diminished excitability of the whole nervous system, which in turn is partly due to fatigue of the afferent nerves, though largely due to the condition of the central nervous system. During sleep we require to apply strong stimuli to produce reflex acts. In the deepest sleep the mental processes seem to be completely in abeyance, so that a person asleep might be compared to an animal with its cerebral hemispheres removed. Towards the approach of the period when a person is about to waken, psychical activity may manifest itself in the form of *dreams,* which differ, however, from normal mental processes. They consist either of impressions, where there is no objective cause, or of voluntary impulses which are not executed, or trains of thought where the reasoning and judging powers are disturbed. Often, especially near the time of waking, the actual stimuli may so act as to give rise to impressions which become mixed with the thoughts of a dream. The pupils are contracted during sleep in proportion to its depth; so that in the deepest sleep they do not become contracted on the application of light. The pupils dilate when sensory or auditory stimuli are applied, and the lighter the sleep the more easily is it accomplished—they are the widest at the moment of awaking.

The soundness of sleep may be determined by the intensity of the sound required to awaken a person. Some observers find that at first sleep deepens very quickly, then more slowly, and the maximum is reached after one hour; it then rapidly lightens, until several hours before waking it is very light. We therefore sleep

soundest the first two hours, and weakest towards morning. The deeper the sleep the longer it lasts.

The cause of sleep is the using up of the potential energy, especially in the central nervous system, which renders a restitution of energy necessary. Perhaps the accumulation of the decomposition products of the nervous activity may also act as a sleep-producer. Sleep cannot be kept up for above a certain time, nor can it be interrupted voluntarily. Many narcotics rapidly produce sleep. *Hypnotics,* such as opium, morphine, potassium bromide, and chloral, are drugs which induce sleep.

Strength and Liminal Intensity of Stimuli.—Homologous stimuli act upon the sensory organs only within certain limits as to strength. Very feeble stimuli at first produce no effect. The strength of stimulus which is just sufficient to cause the first trace of sensation is called by Fechner the " liminal intensity " of the sensation. As the strength of the stimulus increases, so also do the sensations, but the sensations equally increase when the strength of the stimulus increases in relative proportions. Thus, we have the same sensation of equal increase of light when, instead of 10 candles 11, or instead of 100 candles 110 are lighted—the proportion of increase in both cases is equal to one-tenth. As the logarithm of the numbers increases in an equal degree when the numbers increase in the same relative proportion, the law may be expressed thus: " The sensations do not increase with the absolute strength of the stimuli, but nearly as the logarithm of the strength of the stimulus." This is Fechner's " psycho-physical law," but its accuracy has lately been challenged by Hering. It holds good only with regard to stimuli of medium strength. If the specific stimulus be too intense, it gives rise to peculiar painful sensations —e. g., a feeling of blindness or deafness, as the case may be. The sense organs respond to adequate stimuli, but only with certain limits of the stimulus—e. g., the ear responds only to vibrating bodies, emitting a certain range of vibrations per second; the retina responds only to vibrations of the ether between red and violet, but not to the so-called heat vibrations or to the chemically active vibrations. It was Weber who worked out the relation between the intensity of stimuli and the changes in the quantity of the resulting sensations. He used the method of " least observable differences," as applied to sensations of pressure and the measurements of lines by the eye. Hence it is called

CORTEX.

OPTIC THALAMUS.

HORIZONTAL
SECTION OF
CRUS.

CEREBELLUM.

SENSORY
CRANIAL
NERVES.

ANTERO-LATERAL
ASCENDING
TRACT.

HORIZONTAL
SECTION OF
SPINAL CORD.

DIRECT
CEREBELLAR
COLUMN.

POST.
COLUMN

Each cell of the ganglia of
the posterior roots of the spi-
nal cord (or of the cranial
nerve nuclei) gives off two
main processes. One, the den-
drite, goes to the sensory end
organ in the skin or elsewhere,
bringing impressions to the
cell, and constituting the pe-
ripheral sensory nerve-fibre;
the other, the axone, enters
the cord through the poste-
rior root (or runs from the cra-
nial nerve nuclei inward), com-
ing into relation with other
neurones.

SENSORY BRANCH
TO MOTOR CELL
IN ANTERIOR
HORN.

GANGLION OF
POSTERIOR (SEN-
SORY) ROOT.

AXONES.

GANGLION
CELLS.

DENDRITES.

RED = INDIRECT SENSORY PATHWAY.

Fig. 7.—Sensory Pathways. The *direct sensory path* (in blue) (for touch, pain, and
temperature) runs from posterior root across the cord to antero-lateral column, to
tegmentum of crus, to optic thalamus, to cortex. The *indirect sensory paths* (in
red) (for co-ordinative sensations from muscles, joints, and viscera) run upward on
same side, *via* the direct cerebellar tract and the posterior column, decussating at
upper part of cord, to cerebellum, to optic thalamus, to cortex. (From Butler.)
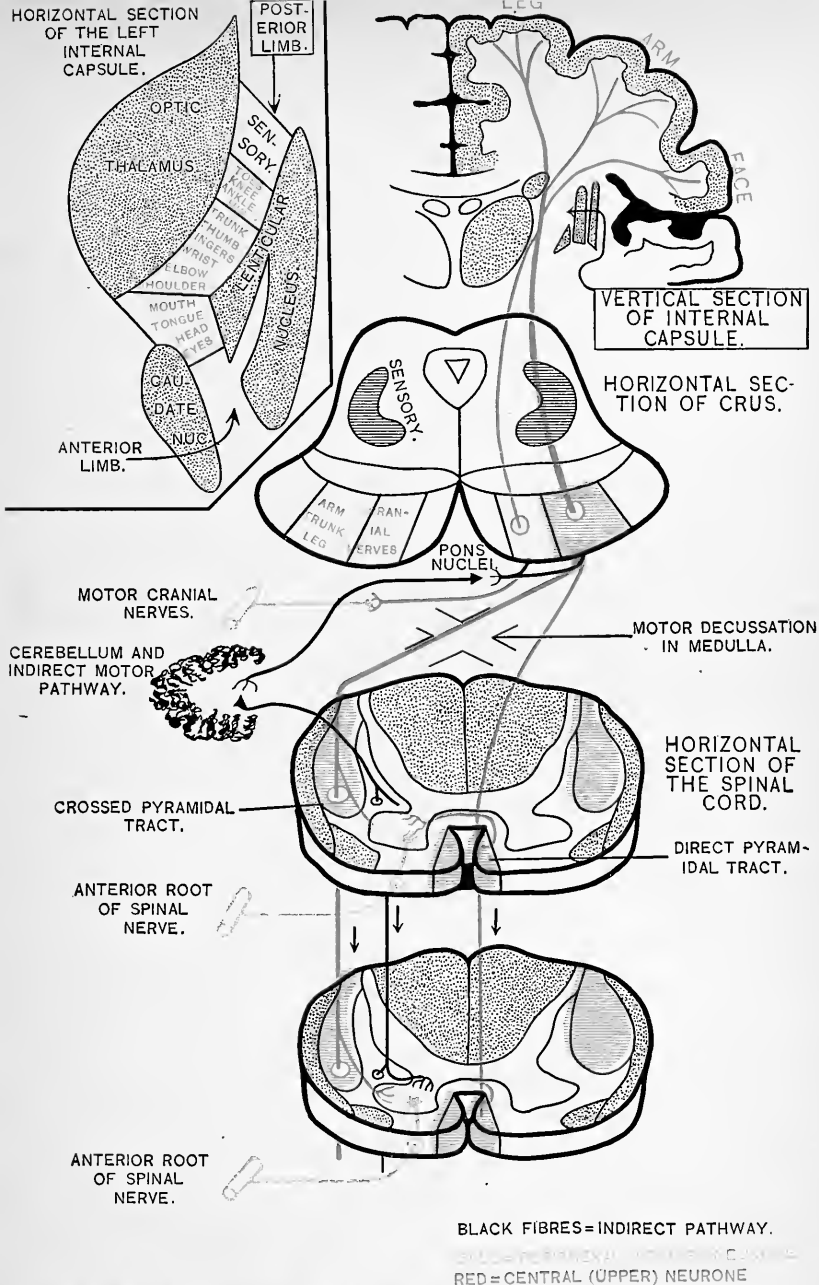
FIG 8.—MOTOR PATHWAYS. *Direct motor path* (in red) (for voluntary impulses, runs from cortex, *via* corona radiata, internal capsule, crus, pons, medulla, crossed and direct pyramidal columns, to motor cells of anterior horn; the cranial nerve motor fibres (in red) cross at various levels in crus, pons, and medulla. *Indirect motor path* (in black) (for muscular co-ordination and higher reflex and automatic movements) runs from cortex to pons nuclei, to cerebellum, to lateral fundamental column, *via* the peduncles, the fibres terminating at various levels in the anterior horn. (From Butler.)

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Weber's law; but Fechner expanded it, and assumed that all just observable differences are equally great, and so the law is sometimes called by his name. Generally speaking, it is called *Weber-Fechner's law.*

After-Sensations.—The term " after-sensation " is applied to the following phenomenon: viz., that, as a rule, the sensation lasts longer than the stimulus producing it; thus there is an after-sensation after pressure is applied to the skin. *Subjective sensations* occur when stimuli due to internal somatic causes excite the nervous apparatus of the sense organ. The highest degrees of these, depending mostly upon pathological stimulation of the sensory cortical centres, are characterized as *hallucinations*—e. g., when a delirious person imagines he sees figures or hears sounds which have no objective reality. In opposition to this condition, the term *illusion* is applied to modifications by the sensorium of sensations actually caused by external objects—e. g., when the rolling of a wagon is mistaken for thunder, etc. In other words, an *illusion* is the misinterpretation of an objective stimulation.

Ideation is the formation of ideas caused by cerebral activity.

The Sympathetic Nervous System: Function, Etc.—It consists of a double chain of ganglia on each side of the spinal column and visceral plexuses. It is chiefly composed of non-medullated nerve-fibres. Its chief function is vaso-motor. Its ganglia are readily paralyzed by *nicotine.*

OTHER NON-NERVOUS TISSUES

Blood-Vessels.—The nerves are richly supplied with blood. The peripheral nerves also are supplied from different arterial branches, but always from one general source. The artery passes to the nerve-sheath obliquely, then divides dichotomously, sending branches for a long distance *up* and *down* the sheath. It may pierce the sheath first, however, then divide as above described. The dichotomous branches send off arterioles and capillaries which form plexuses about the nerve fascicles, the so-called " interfascicular arcades." This division of the arteries subserves the function of preventing large and sudden impact of blood into the parenchyma of the nerves. This resembles also the brain and cord distribution of the blood.

The veins subdivide dichotomously like the arteries, and freely

anastomose with the veins of the muscles, so that muscular action is in close sympathy with nerve function and circulation. Veins of superficial nerves thus also connect with the deep nerve veins.

Lymphatic vessels and spaces are found in the epineurium and perineurium. There are no demonstrable lymphatic vessels in the fasciculi of nerves, but lymph spaces probably exist.

Neuroglia.—In addition to connective tissue, the central nervous system has another substance not found in the peripheral
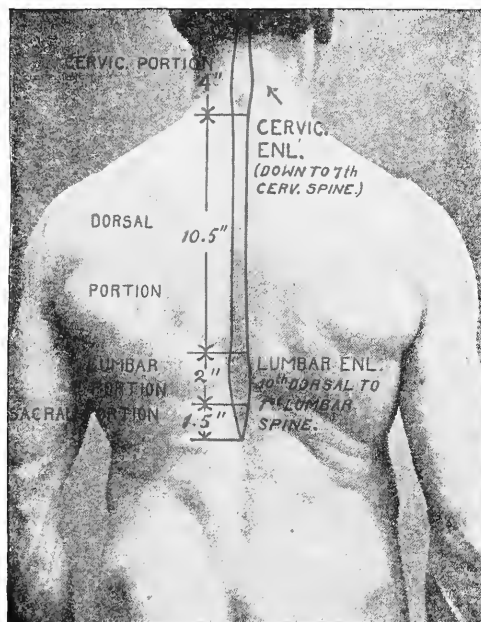
FIG. 9.—Showing the relation of the spinal cord to the dorsal surface of the trunk the relative length of the cervical, dorsal, lumbar, and sacral portions; and the position of the cervical and lumbar enlargements.

nervous system. It is called the neuroglia and is derived from the epiblast. Its cells are very numerous and are finely ramified together, in the which they support the nerve-cells. They are also called "spider cells" and consist of a large nucleus and processes.

SECTION III.—CHEMISTRY

Neuroglia.—One point in the chemistry of the neuroglia should preface this section—i. e., the processes of these cells be-

come charged in their chemical and physical characters so that they take on different stain from that of the cell body itself (Weigert). The scavenger cells of Lewis are the neurogliar cells produced by inflammatory irritation which carry off irritating products; and this must be some form of chemical combination as yet awaiting the physiological chemist to solve.

Nervous Tissue.—According to Krause and Bischoff, the specific gravity of nervous tissue is 1.036; that of the brain 1.038, of the spinal cord and nerves 1.034. The reaction is alkaline owing chiefly to the production of lactic acid.

Water makes up three-fourths of nervous tissue. There is more water in the gray than in the white matter, and least water in the sympathetic nerves. Inorganic salts are about 5 per cent of the total constituents of nerve-tissue. According to Breed, phosphorus is the largest single element, and it is combined with calcium, magnesium, sodium, potassium, and iron-forming phosphate salts. Chloride of potassium is the most important of the other constituents.

Table of Chemical Constituents of Nervous System

(Baumstärk—quoted by Hammerstein and by Dana)

	White matter.	Gray matter.
Water in 1,000 parts	603.35	769.97
Solids " " "	304.65	230.03
Protagon { cerebrin / lecithin (neurin) }	25.11	10.08
Insoluble albumin and connective tissue	50.02	60.79
Cholesterin	45.12	23.81
Nuclein	2.94	1.99
Neurokeratin	18.93	10.43
Inorganic salts	5.23	5.62

Protagon is a complex substance of fatty matter containing nitrogen, united with glycerin-phosphoric acid instead of glycerin. This protagon is made up of two bodies, *cerebrin* and *lecithin,* the latter of which contains an ammonia compound called neurin. Protagon is especially abundant in the white matter. The gray matter contains nuclein, important in cell metabolism. Nuclein ($C_{29}H_{49}N_{9}P_{3}O_{22}$ Miescher) consists of nucleic acid, a substance rich in phosphorus and a various amount of albumin. The nerve-cells (gray matter) also contain various amounts of albumi-

nous substances. According to Halliburton, *nuclein* and its con-
gener *nucleo-albumin* are designated *albuminoids*. They both con-
tain albumin and are found chiefly in the nucleus. The albu-
minous substances are also called proteids and contain little or no
phosphorus and make up most of the cytoplasm or cell body.

CHAPTER II

GENERAL PATHOLOGY

THE nervous system, composed as it is of nerve-cells and fibres forming the neurones, the connective tissue, the neuroglia, blood-vessels, and lymphatics, must have in its pathology disease of one or many of these structures. Most diseases affect the blood-vessels, connective tissue, or neuroglia primarily, and parenchyma secondarily, although in many affections it still remains doubtful which is first diseased.

Forms of disease which affect the nervous system are, therefore, as follows:

1. Functional and nutritive disorders, including those of metabolic and glandular defects as in exophthalmic goître, cretinism, or myxœdema and acromegaly.

2. Malformations, lack of proper development or agenesis; defective development or dysgenesis.

3. Anæmia, hæmorrhage, and arterio-venous disease.

4. Atrophy, degeneration, softening, gliosis, and sclerosis.

5. Hyperæmia, œdema, and inflammations.

6. Tumours, parasites (echinococcus, etc.).

7. Syphilis, tuberculosis.

Under *nutritive* and *functional disorders* of the nervous system come defects in metabolism, as uric-acid diathesis causative of a certain number of cases of neurasthenia; also under this comes, as just mentioned, other autochthonous poisons such as leucomaines. Then come the extrinsic poisons, as ptomaines developed from intestinal intoxication, etc.

Sclerosis is a process of connective-tissue proliferation in which the normal parenchymatous tissue is supplanted by connective tissue. This is the *result* of degeneration (next to be considered). Neuroglia overgrowth also usually occurs in sclerosis.

Degenerations may be divided as follows, into *acute* and *chronic*; and these again are subdivided into *primary* and *secondary*

and *mixed*. Under *primary* come the progressive muscular atrophies of spinal origin, myelomalacia and tabes dorsalis, or any of the system diseases of the cord. Under *secondary* degenerations, we may give as examples secondary lateral sclerosis as in that following upon hemiplegia of cerebral origin or in degeneration (diffuse) of the cord following chronic myelitis or syringomyelia. Acute degeneration usually brings about a *softening* or *necrosis*. If repair occurs a *cicatrix* is formed.

Primary degenerations are, of course, due to inherent defect in nutrition or to some poison (as that of syphilis in the fourth stage) acting directly on the fibre or cell. Secondary degeneration is *per contra* due to separation of the nerve-fibre or cell from its trophic centre, or to cutting off of its vascular supply, or to injury as in pressure neuritis, etc.

The most usual extrinsic poisons that cause degenerations are arsenic, lead, phosphorus, or the poisons of infectious diseases as of la grippe. Endarteritis and arteriosclerosis of old age also cause degenerations by obliterating the lumen of vessels, and thus preventing proper nutrition of the part beyond. There is a legion of causes which with all our scientific advancement are as yet unknown, but are productive of premature death of the cell. Whether certain scleroses are forms of proliferative inflammation is as yet also a debated question by pathologists, although the tendency is now to term the so-styled chronic inflammations as really degenerative processes, and that, as indicated above, the primary trouble is in the parenchyma.

Gliosis.—Dèjerine is the leader of the French school who believes that some of the chronic degenerative diseases are due to proliferation of neuroglia. This overgrowth of neurogliar tissue rather than connective tissue is termed *gliosis*.

Inflammations.—Since the pathology of most of the types of disease above outlined are given under their respective heads, we shall dwell upon the main pathologic changes only in this chapter.

Inflammation should be understood as to its nature, as the student should also have a clear comprehension of *degeneration* of nerve-tissue. Inflammation has primarily to do with blood-vessels, then lymphatics, and finally formation of connective tissue. It is the reaction of an organism to an irritant, so that wherever there is inflammation there has been irritation. Products of growths of micro-organisms, or some irritant the product of tissue change, are

usually the immediate causes of the irritation. But certain chemical substances, as alcohol, lead, or arsenic, as previously mentioned in this section, *may* produce inflammations directly; although they are more apt to be causative of primary or degenerative (destructive) processes.

When the irritant is removed, inflammation tends to subside— i. e., it is then *regressive* not a *progressive* process.

Inflammations are further divided into—

1. Productive forms (Delafield).

2. Exudative forms—these forms may be simple inflammations, without necrosis or with necrosis, purulent or purulent and necrotic.

Productive or proliferative inflammation is a process in which there is little congestion and exudation, but new connective tissue *slowly* forms. It is usually chronic. Examples of this form are syphilitic or tubercular inflammations (producing specific granulomata or tubercles). Lead, alcohol, arsenic may also produce this form, as may the poisons of rheumatism, gout, diabetes, states of inanition (*pseudo*-palsies of rickets, etc.).

Exudative inflammation is associated with first a congestion, then stasis, exudation of white blood corpuscles (amœboid movement), diapedesis of the red blood cells, transudation of blood serum, the final formation of fibrin; the complete result being an exudate containing white blood cells, now called pus cells, and fibrin. In some instances there is no destruction of tissue in this form of inflammation, and after its subsidence the tissues affected return to the normal state. In other cases the nerve tissue is partly destroyed. In the *purulent* form there is great increase of pus cells and but little fibrin. If the tissue is also destroyed it is a *purulent* and *necrotic* form of inflammation. In some forms of exudative inflammation increase of connective tissue takes place from the outset, and this latter continues until the inflammation subsides. Most forms of this exudative type are acute or subacute. *Inflammatory* œdema, so called, is of this type of inflammation.

Nerve-cells proper once destroyed never re-develop. This is not true of nerve-fibres, and while these frequently develop again, they but seldom do so within the tracts of the central nervous system. The peripheral nerves when *regenerating* always grow *from* their trophic centre. Nerve-tissue in *brain*, *cord*, or *nerve* will not reunite, after section, by direct union. With but very few excep-

tions there is a primary *degeneration* of the cut ends, which is then followed by *regeneration*. Nervous tissue is further dependent upon its blood supply and its *trophic* connection for its vitality. The nerve-cell is absolutely dependent on the blood supply, while the neuraxone is dependent upon the trophic influence of its connecting cell, though it can survive for a time the simple exclusion of its blood supply. If the neuraxone is injured it affects the cell, which can be repaired, however.

Tumours will be described under the heading of Neoplasms.

CHAPTER III

NOMENCLATURE IN NERVOUS DISEASES

Neurosis applies to a functional condition of the nervous system, wherein the higher centres (of the mind) are not involved, such as in neurasthenia.

Where the higher faculties of mind are involved the name *psychosis* is given, as in hysteria or insanity.

Stupor is that state of unconsciousness in which the patient can be partially or completely aroused; and in which the reflexes are preserved.

Coma is that state of unconsciousness from which the patient cannot be aroused and wherein the reflexes are absent.

Hyperæsthesia is a term used to indicate increased sensibility.

Hypæsthesia applies to decrease of sensation.

Paræsthesia is a perversion of common sensation, such as tingling, numbness, or *formication,* which latter is a sensation as though ants were crawling over the body.

Spasm or *convulsion* is a symptom in which there is violent contraction of the muscles, and may be *localized* or *general.* Frequently spasm is used in the sense of a localized convulsion alone, although this is not technically correct, and local spasm is properly called " tic." [1] such as " tic " of the face—a spasm of the seventh nerve. Convulsions are further divided into *clonic* and *tonic.* In the former the movements are in rapid succession, and in the latter the contraction is maintained for a long period of time. Hysterical convulsions are particularly apt to be of a tonic nature. (See illustration, p. 56.)

There are certain definite affections of different tracts of the central nervous system which are given special prominence in the

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[1] *Tic* may also apply to a painful paroxysm in neurological nomenclature; but then the prefix *painful* is used in explanation, or an affix as in "*tic doulou-reux.*"

No. of Case. Name of Book. Date. Service of Doctor.

Name. Residence. Age. Sex. Race.

General Statement to Aid in Classifying Case.

Family History:

>Hereditary Tendencies—Health of Parents—Syphilis—Gout—Diabetes, etc.

Personal History:

>Married or Single—Children—Relation of Work to Present Trouble—Illnesses — Injuries — Syphilis — Nervous Diseases—Habits—Temperament—Life, Active or Sedentary—Occupation—Exposure to Poisons—Malaria — Lead — Tobacco — Alcohol—Narcotics—Opium, etc.—Usual Weight—Height.

Date of Onset of Present Trouble:

>Supposed Cause—Mode of Onset and Outline of Course.

General Aspect:

>Weight now—Colour—Skin—Hair—Eyes—Scars on Head, etc.

State of Organs:

>Present—Comparing Past.

1. Digestion:

>Tongue—Bowels—Appetite—Rectum—Teeth—Liver—Spleen—Taste.

2. Respiration:

>Lungs—Throat—Nose—Smell.

3. Circulation:

>Heart—Pulse—Arterial Tension—Blood—Œdema.

4. Uro-Genitory:

>Catamenia — Leucorrhœa — Displacements — Phimosis Scars—Sexual Functions — Power, etc. —Urinalysis — Colour — Sp. Gr.—Albumen—Sugar—Uric Acid—Oxalates—Urates.

5. Nervous System:

>(a) Motility—Voluntary Movements—Strength—Grasp—Co-ordination—Spasms—Reflex Capacity—Knee-Jerk—Arm-Jerk—Jaw-Jerk—Skin—Cremaster, etc.—Muscle-Jerks—Clonus—Station—Sway—Bladder Control.
>(b) Sensibility—Subjective Sensations—Hyperæsthesia—Anæsthesia, etc.—To contact (Æsthesiometer), to pain, to temperature—Intellectual Functions—Hallucinations—Delusions—State of Will—Predominant Ideas—Specify Insane Acts—Sleep—Vertigo—Tenderness over Nerves—Headaches—Electrical Examination—Trophic Alterations—Vaso-motor Activities.

6. Sight:

>Vision (Type—Fingers—Colours)—Field — Fundus—Ocular Muscles—Pupils.

7. Hearing:

>Deafness—Wax—Tinnitus.

55

study of diseases of the nervous system, and lead to assist in the
diagnosis through knowing the results of anatomical or physiolog-
ical perversions in them. In the sensory tracts we have mani-
fested pain, dull or shooting in character; *parœsthesia,* or numb-
ness of a part; *causalgia,* or burning pain, frequently occurring
in peripheral neuritis—all due to irritative lesions of these tracts.
A diminution of the function of the sensory tracts will produce

Fig. 10.—Position of Hand in Local Tonic Hysterical Spasm

hypæsthesia, anæsthesia.
Different distributions of
anæsthesia are of *nerve-
trunk,* the *circumscribed,*
the *segmental* or *glove-like*
—where the anæsthesia ex-
tends up an entire extrem-
ity, such as from the finger-
tips to the elbow, as in
cases of hysteria. *Hemian-
æsthesia* is a condition
where half of the body is in-
volved—this being of two
different kinds — organic
and functional, a point of
difference being that func-
tional or hysterical anæs-
thesia, so called, has a dis-
tinct dividing line in the
median line of the body—
whereas organic hemianæs-
thesia will extend slightly
beyond the median line, due
to the distal crossing of

nerve-fibres beyond the median line; or, better, this latter is anæs-
thesia from the centre to the ultimate end of the nerve-twigs. The
destructive lesion of the spinal cord involving one-half of it will
produce symptoms of complete hemianæsthesia and slight motor
loss below the site of lesion on the opposite side, with complete
motor loss on the side of lesion. Usually it is only pain
and temperature sense that are lost, since the lesion is, as a rule,
limited. This is called Brown-Séquard paralysis, named for
him who first described it, and is due to the fact that the sen-

sory fibres upon entering the cord cross to the opposite side, then pass up.

In *pontine lesions* we may have a hemianæsthesia of same side of the face with motor and sensory palsy on the opposite side of the body to the lesion.

Algesia refers to the pain produced in a part either by external or subjective stimulation of sensory nerve-fibres or neurones.

Muscle sense is that phenomenon by which we distinguish space, shape, size; also the location of parts of the body.

Asteriognosis refers to the inability to appreciate dimensions, density, and shapes of objects.
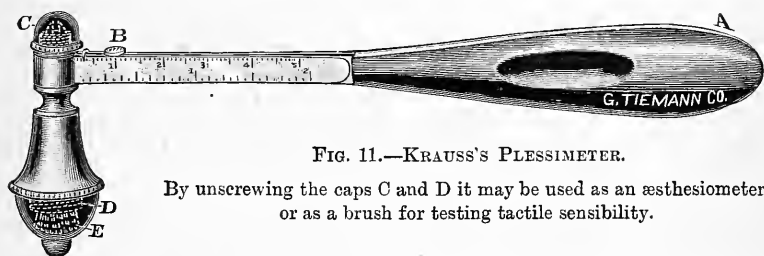
FIG. 11.—KRAUSS'S PLESSIMETER.

By unscrewing the caps C and D it may be used as an æsthesiometer, or as a brush for testing tactile sensibility.

Temperature sense is the faculty of appreciating temperatures of bodies, such as heat and cold.

Reflexes are phenomena produced by irritation or by stimulants of some sort either from within or from without. Ordinarily, the reflex consists of an *afferent* impulse, which is carried over the sensory tract to the nerve-centre in the cord or brain. The impulse is then transmitted to the *centre*, which consists of a collection of nerve-cells within the cord or brain; and finally the *efferent* impulse completing the reflex is carried over the motor tract or fibres from the nerve-centre, producing motion, secretion, and various physiological phenomena of the body, depending upon the nature of the reflex action.

Reflexes are divided into *superficial* and *deep* and *visceral*. Superficial are those occurring upon the surface of the body, such as skin and mucous membranes. We have among them the *pupillary skin* reflex, which consists of a dilatation of a pupil, produced by the irritation of the skin of the neck, as in pinching the same, the impulse being carried up by the cervical sensory nerves, and down as a motor impulse through the motor sympathetic nerve-fibres to the dilator muscles of the pupil. The *pectoral* reflex con-

sists in contraction of the pectoral muscles through the skin over them. The *epigastric* reflex consists in a contraction in the epigastric region through the fibres of the recti muscles. The *abdominal* reflex consists of contraction of the abdominal muscles when the abdomen is irritated. The *supra-orbital* reflex of McCarthy consists in elevation of the lower lid when the supra-orbital region is gently tapped. There are a number of others not of practical importance to the student.

FIG. 12.—SHOWING THE JENDRASSIK METHOD OF OBTAINING THE KNEE-JERK.

The *cremasteric* reflex consists in retraction of the testicle when the skin of thigh is irritated.

The *plantar* reflex consists of flexion of the toes when sole of the foot is irritated.

The *palmar* reflex consists in jerking of hand and closing of the fingers upon irritating the skin.

The *infra-orbital* reflex consists of the elevation of the lower eyelid when the supra-orbital region is irritated by a blow.

Deep reflexes or *muscle reflexes* are produced by tapping a muscle or tendon and causing contraction of the muscle. The knee-jerk is produced by tapping the ligamentum patella, the leg being loosely *pendant* at right angles to the thigh. There is extension of the leg.

The *tendo-Achilles* reflex is produced by tapping the tendo-Achilles, when extension of the foot will occur, due to the contraction of the gastrocnemius group of muscles.

The *contra-lateral* or *adductor femoris* reflex is one produced by tapping the ligamentum patella on one side, the patient sitting on the edge of a chair with both limbs loosely resting on the feet. If this reflex is present adduction of the opposite thigh will occur. This is an inconstant reflex, and although it cannot be said to be pathological in significance, it is much increased in cases of spastic disease, as a rule, especially when involving the lateral columns of the cord.

Ankle clonus is a deep reflex, always pathological, and is produced by sudden flexion of the ankle, the physician support-
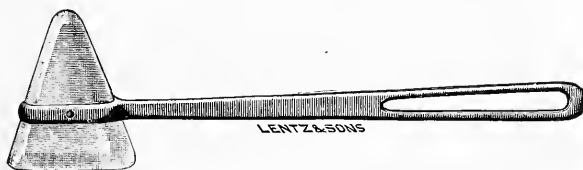
FIG. 13.—TAYLOR KNEE-JERK HAMMER.

ing the calf with one hand and firmly pressing on the sole or ball of the foot quickly. If present there will be a to-and-fro clonic involvement of the foot due to contraction of the soleus [1] and gastro-intestinal muscle. This is called true ankle clonus.

A *psuedo-ankle* clonus is that inconstant clonic contraction of the same muscle group, occurring in neurasthenia or hysteria. It is also called abortive ankle clonus, since it is soon exhausted. It does not signify organic disease of the cord, as the true form always does.

The *elbow-jerk* consists in the extension of the elbow, produced by tapping the tendon of the triceps muscle when the arm is held pendant over the table or other object.

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[1] Weir-Mitchell believes it is entirely due to action of the soleus.

The *biceps-jerk* consists in flexion of the elbow, when the biceps tendon or muscle is suddenly tapped.

The *extensor* reflex of the wrist consists of the extension at the wrist when the extensor group of muscles are tapped.

The *wrist* reflex proper consists in flexion when the flexor muscles are tapped.

Fig. 14.—Showing Method of Obtaining the Triceps and Supinator Jerk.

The *jaw-jerk* consists in sudden closing of the mouth when tapping over the mental process of the inferior maxilla.

Paradoxical contraction consists of a tonic contraction of the anterior tibial muscles, produced by a sudden flexion of the foot or the leg. This sudden shortening of the muscles indicated causes the tonic spasm which is always pathological.

Superficial and *deep* reflexes are said to be increased when there

is a decided increase of the movement over the normal; lessened when there is a lessening of the normal amount of movement upon its development. Of course reflexes are absent where no response occurs. The two extremes of reflex action are *spastic* where the reflexes are greatly increased, and *absent* when there is no reflex at all produced on external stimulation. Several other terms are used in describing reflexes, such as moderately increased and greatly increased, and related to the spastic as described. Also the other extreme—slightly diminished and greatly diminished, exists before abolition of the reflexes occurs.

Visceral reflexes is the third group, and consists of the action of the various viscera, etc., induced by irritative phenomena; as the peristaltic movements of the stomach, after taking of food.

The Eye.—The *light* reflex is produced by throwing a bright light into the eye, causing contraction of the pupil. *Accommodation* reflex is brought out by causing the subject to look at a near object when the pupil also suddenly contracts. When the light reflex is lost and accommodation is still present the Argyll-Robertson pupil is said to exist.

Reflexes of the bladder, rectum, and sexual apparatus are also important; the two former being under control of the higher centres. If the tone of the compressor urethræ is intact, the reflex being lost, we have incontinence of overflow as a sign.

Damage to the *genital* reflex arc causes loss of erection and sexual desire. Loss of inhibition may cause priapism.

TROPHIC, VASO-MOTOR AND SECRETORY PHENOMENA

By *trophic disturbances* we refer to certain conditions, as the wasting of muscles, false hypertrophy of muscles, bedsores, joint disease, trophic changes in same, such as the arthropathies, brittleness of the nails, hypertrophy of bone, such as in Paget's disease, where the cranial bones are enlarged.

Vaso-motor disturbances refer to the conditions dependent upon disease of the nervous system, as flushing, œdema, angeioneurotic œdema, coldness and clamminess of the extremities.

Secretory phenomena and disturbances thereof, consists of, among others, retention, or too much elimination of certain secretions; in the former in the case of the thyreoid gland, we have

developed a disease to be studied in another chapter—cretinism
and myxœdema. *Excess* of thyroid secretion probably occurs in
exophthalmic goître.

EXAMINATION OF THE PATIENT

Examination of the patient for
nervous disease includes a study the
most thorough in general medicine, in
order to exclude other maladies; and
specifically, of course, for the deter-
mination of the status of the nervous
system. It will be in keeping with the
book to pass by intricate medical meth-
ods, therefore, insisting upon their im-
portance, however, and to hasten on to
the examination of the nervous system
itself.

Gait and Station.—*Station* is the
attitude, the manner of holding the
body in the upright posture, the feet
being in the position of "atten-
tion."

Fig. 15. — WEIR MITCHELL'S
APPARATUS FOR CLINICAL OB-
SERVATIONS OF STATION.

The station is said to be normal when the patient but
slightly deviates from the erect posture while standing, the
sway tending towards no particular direction. The station is
said to be *abnormal* when the patient does sway markedly.

FIG. 16.—FOOT DYNAMOMETER.

In designating this symp-
tom, right, left, forward or
backward are used to indi-
cate the direction of the
abnormal deviating move-
ment. An unsteady sta-
tion is called *Romberg's
sign*, particularly found in
tabes.
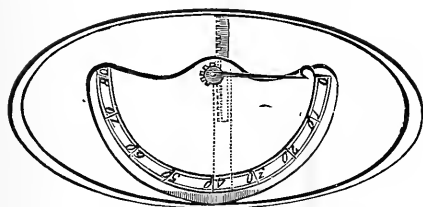
The sway should also be tested with the eyes open and with the
eyes closed, since it will always be found more perverted when the
eyes are shut, especially in diseases where muscular sense is much
disturbed, as in tabes, referred to above.

Gait is the manner of progression of the individual. It is to be noted that there is a difference between the normal gaits of the two sexes. The female, due to the breadth of the hips, has a more or less waddling progression, whereas the male movement is more directly forward in walking.

Patients should not deviate from the direct line of progression in any one direction more than another, although a natural sway does occur, and there are particular family types of normal progression.

Grasp is determined by the dynamometer. It is of use to compare palsy of the hand and forearm muscles.

PATHOLOGIC GAITS

1. **Hemiplegic,** or that type in which the individual carries one side of the body forward with the muscular action of the unaffected side, plus gravity. The patient brings the affected leg
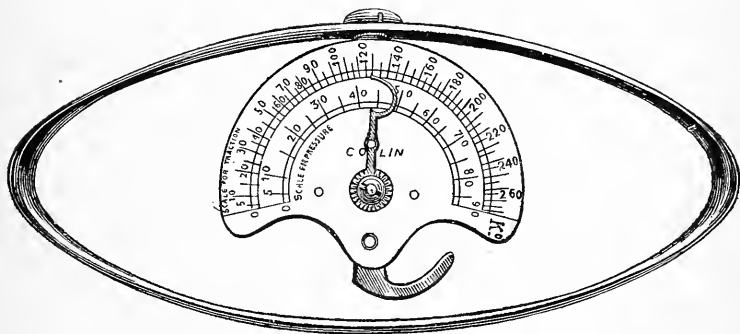
FIG. 17.—HAND DYNAMOMETER.

forward in flail-like fashion, using the opposite foot as a pivot, the toe of the affected side dropping to the ground before the step is completed, so that a tracing of this gait will present a short stride of the affected side, followed by the longer stride of the healthy limb.

2. **Spastic** is one characterized by the forcing of the toes downward in flexion during progression. In its incipiency the wearing out of the toe of the shoe may be the most prominent sign. Later the ball of the foot is " dug " into the floor, the patient stumbling over the slightest impediment. The knee-jerk is found to be in-

creased, though it may be difficult to elicit on account of flexor contractions.

3. **Ataxic** is one where the patient throws the limb outward, forward, and downward, the heel dropping first, and all the movements being inco-ordinate. This gait is decidedly worse when the patient is in the dark or when closing his eyes. The knee-jerk is usually absent. It is increased in ataxic-paraplegia.

4. **Titubation** is characterized by a rather sudden deviation to right or left during progression. As a rule, the patient " catches " himself before more than one or two steps away from the normal are made. This gait is rather characteristic of the inco-ordination produced by cerebellar disease where centres for balance lie.

5. **Steppage gait** is that in which the patient lifts the toes high from the surface, such as normally is done in ascending stairs. The cause for this exaggeration lies in the fact that there is palsy of the anterior muscles of the leg. This gait occurs in neuritis and the muscular dystrophies. It is almost pathognomonic of these conditions.

6. **Festinating gait** (or running gait). Here the patient tends to go forward rapidly, as in paralysis agitans, in which disease a sensation of falling forward (propulsion) or backward (retropulsion) may also occur.

PHYSIOGNOMY

Physiognomy of the patient has much to do with the determination of certain features of the nervous malady. Besides the final delineations of feature, showing depressive mental states or the opposite, a temperament is shown by the examination of the facies, of which the following are of some worth in studying the case—namely:

Lymphatic temperament, in which the face, along with other structures, presents a pale and waxy sallow aspect, and in which the subcutaneous tissues are flabby and the skin is unduly wrinkled. Such persons are apt to be large of skeleton, out of proportion to the musculature, and their circulation is sluggish.

Sanguineous temperament is characterized by the face presenting a florid complexion. Prominence and tension of the subcutaneous tissues, with perhaps excessive development of fat

cells, is seen; the neck usually appearing short and the chest broad, out of proportion to the general physique. It should be stated that this type is predisposed to apoplexy among nervous diseases.

Bilious temperament is, typically, found in those with dark hair and complexion. The salient features are tendency to jaundice, to a melancholy physiognomy, the tongue being usually coated and bowels constipated. Such types are likely subject to nervous depressive diseases, such as hypochondriasis and melancholia, or to the neurasthenias dependent upon gastro-intestinal auto-intoxication in some measure.

Nervous Temperament.—Here the individual expression is one of vivacity, eyes are alert, movements quick; with a musculature that is not large, but the muscles are distinctly outlined, as in the thoroughbred, the bellies being prominent. These people are most likely to come under the list of nervous patients, because they do the world's work.

Neuro-Bilious Temperament.—This is a type we can well designate in America, since our methods of life conduce to modification of the other temperaments towards the nervous; and since the bilious temperament, as indicated, is not itself an important factor in nervous disease, but when added to by the nervous element there is reason to make the above designation. This type is the most difficult to manage in any case of nervous or mental disease.

DECUBITUS

In neurology the position of the patient, as he lies in bed, is important to note in making an examination.

In decubitus of meningitis, as a rule, the patient lies supine, or on his side, with retraction of the head and hyperextension of the back, plus general muscular rigidity.

In hemiplegia the patient usually lies upon the back, the one side of the body being assisted by the normal extremities.

In tetanus and hydrophobia the decubitus is not consistent, but is somewhat like that of meningitis, the patient more frequently remaining on the side, however. Tonic and clonic convulsions occur spontaneously or with the slightest reflex excitement. In both of these diseases there is frothing at the mouth.

Decubitus of an epileptic attack consists of the patient lying on

the back usually in the typical convulsion, with conjugate devia-
tion of the eyes and dilated pupils. Following the attack the
patient remains limp, supine. He may, of course, fall upon his
face in exceptional instances.

Decubitus of neuritis is not constant, depending upon the
degree and extent of nervous inflammation and palsy. The pa-
tient always tends to protect the extremity or part affected, and
this will extend to the guarding of the extremity from any injury
or contact, as of the bed-clothing, when there is active neuritis.

EXAMINATION FOR SENSATION

This is done by means of the æsthesiometer, to determine the
presence, absence (anæsthesia), or diminution (hypæsthesia), or
exaggeration (hyperæsthesia) of common sensations. Dysæsthesia
is an abnormal sensation, such as a feeling of discomfort pro-
duced by ordinary tactile or painful impres-
sion. Paræsthesia is a subjective sensation,
such as a feeling of pins and needles prick-
ing the affected part, and occurs frequently
in early neuritis, as in pressure palsy.

Causalgia is a "burning" sensation
found in some cases of neuritis of chronic
type, as in erythromelalgia.

Formication is the sensation as of ants
crawling over the surface of the skin; often
a delusion of mental disease.

For the finest determination of sense of
touch, a piece of cotton twirled at the end
will be found efficacious; or better the æs-
thesiometer. In testing, it should always be
done symmetrically, since one person may
differ from another, according to the tem-

FIG. 18.—CARROLL'S
ÆSTHESIOMETER.

perament, so that a standard cannot be set
for all persons from one case. It should be
remembered, too, that special parts of the body are more sensitive
than others, such as the face and the lips, tongue, and palms of the
hands and feet.

To test the *thermic sense,* heat and cold are applied through
water in test-tubes. The absence of thermic sense (thermo-anæs-

thesia) is to be expected in cases of syringomyelia, where the touch sense may be retained in the absence of thermic sensation— the so-called *disassociation of sensation.*

Algesia or pain sense is tested by means of a sharp instrument, as the æsthesiometer. This, also, may be absent (analgesia) with preservation of the other forms of sensation in some cases of hysteria, etc.

EXAMINATION FOR MOTION

The motor phenomena are examined for, first, as to palsies. These can be tested by means of the hand or foot dynamometer, which is an instrument by which record is made on a dial of the amount of force used to compress the oval spring. In testing the strength of the lower extremity, the instrument is suspended from the shoulder, and by means of a strap, attached also to the dynamometer, the amount of pressure can be registered. This should be used bilaterally, since the record is only for comparison. The palsy in other parts is noted by the strength shown in movement of the part of the muscles, as in effort to lift some object, etc. Individual muscle palsies are noted by observation, the anatomical position of the part involved, and in relation to the motor nerve supply, thus giving way to opposing normal muscles.

The opposite of paralyses, *convulsions*, are determined by means of the sense of sight.

A *contraction* is a shortening of the muscle of a part, which cannot be forcibly extended even under etherization of the patient.

A *contracture* is the shortening of a muscle, which can be relieved by firm extension, the muscle therefore not being actually organically diseased. It may be necessary to etherize the patient to accurately test this.

Paralysis may be *partial* or *complete*, the former scientifically being designated *paresis.*

The forms of paralysis are:

Hemiplegia, involving one-half of the body.

Monoplegia, where the palsy is confined to one limb of the body.

Paraplegia, where both the lower extremities are affected.

Diaplegia, where the palsy involves corresponding extremities, as the two legs, two arms, or all four limbs.

Loss of the special sensibility of the muscles, articular sur-
faces, and tendons produces ataxic movements.

Ataxic movements are inco-ordinate movements, as in loco-
motor ataxia; or in cerebellar disease or in neuritis.   Apprecia-
tion of weight and position of limbs are lost in ataxia.

Static ataxia consists in loss of equilibrium when the patient
or part is at rest.

Cerebellar ataxia is produced by cerebellar disease.

Motion of an unparalyzed limb or a limb that is partially
paralyzed will occasionally produce movements in the correspond-
ing paralyzed extremity.   This is designated as *associated move-
ment.*   A forced *movement* is one produced in spite of the patient's
will, as when the patient is suddenly whirled about in various
directions.

Some other abnormal movements are tremors, spasms, and
choreiform convulsions.

Tremors are fine, coarse, intentional, and continual.

The following table gives the various causes of *tremor* and
their character:

CAUSE.		Type of tremor.	Rapidity.
Toxic...	Arsenic. Lead. Alcohol. Tobacco. Tea. Coffee.	Intention in early stages; later may become con-stant; may then be in-creased by exertion.	Rapid.
Neuroses	Hysteria. Neurasthenia. Exophthalmic Goître.	Ibid.	That of hysteria some-times is slow; others always rapid.
Senility		Ibid.	Rapid.
Heredity		Ibid.	Rapid.
Any disease of brain, spinal cord, or periphe-ral nerves, excepting multiple sclerosis and paralysis agitans.		Ibid.	May be slow or rapid, or both combined.
Paralysis agitans........		Often ceases for a few seconds after muscular exertion.	Slow.
Multiple sclerosis.........		Intention only.	Slow.

Spasms may be *tonic* or *clonic,* the latter consisting in rapid
movements; but when the contraction persists it is designated
tonic; when permanently persisting, it produces what is called
muscular *rigidity,* as in the extremities, neck, or trunk.

Choreic movements are sudden, or jerking, irregular inco-ordinate movements.

Convulsive tic is a form of choreic movement confined to certain groups of muscles and limited to muscles physiologically grouped for certain functions, as the respiration, or expression, or locomotor.

Athetosis is the vermicular movement described first by Hammond, observed in cerebral disease, as in porencephaly.

Conjugate deviation is a phenomenon that is frequently present in diseases of one side of the brain. In this the eyes are directed *towards* one side (of lesion), with or without the corresponding rotation of the head.

If the lesion is a simple irritative one the deviation is away from the side of the lesion. The first is paralytic, the latter spasmodic.

PRESSURE SENSE

Pressure sense is determined by means of weights, alternating one with the other, until the power of discrimination is nicely met with the least amount of weight. Differences of temperature should be excluded, also the prevention of the displacement of the weights in this test, and the area or part tested should be held in relaxed position. For making the finer tests bits of cork are used.

MUSCULAR SENSIBILITY

This test depends somewhat upon idiosyncrasy of the individual. To exclude surface differences in testing by balancing weights, they can be suspended in a towel or by cords. By repeated experiments, it is thus possible to determine as between the cutaneous sensibility (which will interfere with the test to the extent of two or three ounces pressure) and the muscular sense.

The pressure sense is a composite physiological phenomenon, consisting of the sense of coarse movements of the limb, the posture sense and the pressure sense.

SENSE OF THE APPLICATION OF WEIGHT

This is determined by the piesmeter of Beard and Rockwell, which instrument is made up of a cylinder, $\frac{3}{4}$ of an inch in diameter and 3 inches long, in which is a piston kept pressed back to

its fullest extent by a spring. At the end of the piston rod is a flat disk which is pressed against the skin. The physician then presses against the end of the cylinder, forcing the piston into the barrel, and the amount of pressure made before it is recognised by the patient is indicated by a scale on the instrument. In this connection it should be stated that there is a similar instrument, which is called the algometer, devised by Dr. Arthur MacDonald, by which pain is tested in a similar manner to that of the above instrument. Instead of the plain disk, however, a sharpened point is forced upon the part to be tested.

SENSATIONS OF MOTION

It may be necessary to diagnosticate diseases of the semi-circular canals. the vestibular nerve and its terminations, from affections of the cochlear nerve, which latter is the true nerve of hearing. Since the two nerves are so closely approximated in most deaf persons, it will be found that sensations of motion are also affected in them. So that it is chiefly where the periphery of the vestibular nerve is affected, the patient not being deaf, there-fore, that abnormal sensation of motion are produced in hearing individuals. In this test, deafness or not should, of course, be first determined. Then rapidly rotate the table upon which the patient stands, the top of which is placed upon a pivot, as suggested by Sanford. In health the direction of rotation can be recognised when the rate is as low as two degrees per second. or even lower. In some deaf-mutes the sense of motion is entirely absent. This latter is probably a reason why deaf people seldom get seasick.

OCULAR DISTURBANCES

In this we examine for changes in the optic nerve, such as neuritis. atrophy. occurring in brain tumour. meningitis. etc.; also for changes in the retina, especially concerned in specific disease of the nervous system. Refractive errors are not a part of neuro-logical examination. Nystagmus or involuntary oscillation of the eyes, usually bilateral and horizontal. is present in affections of the cerebellum and in disseminated scleroses. It is irregular or rotary in type in blind persons. Nystagmus may also be congenital.

Hemianopsia is loss of one side of the visual field. whereas *hemiopia* refers to loss of visual power in one-half of the retina.

The latter term is generally used in describing peripheral disturbances, whereas hemianopsia is used in the study of brain diseases. The lesion causing hemianopsia is situated anywhere from the cortical centres of vision in the occipital lobe to the optic chiasm. In rare cases it may be necessary to study areas of obscuration of vision, as quadrants or irregular areas. Hemianopsia may be vertical, horizontal, bilateral, binocular, or lateral homonymous.

Homonymous hemianopsia indicates blindness of the *inner* half of one field and the *outer* half of the other field of vision.

Amblyopia indicates dimness of vision.

Amaurosis indicates total blindness.

Hemianopsia can be roughly determined by having the patient look at a fixed point, then by moving an object from without in, in all portions of the visual field, when, if it is not present, the moving object will be perceived at the proper limit. The perimeter of Emerson is also used by the ophthalmologist to make these tests.

Errors of refraction and exophthalmos should also be noted.

Wernicke's hemiopic pupillary inaction consists in lack of contraction of the iris when a ray of light is thrown on the blinded side of the retina. Since the reflex centre of this arc is located in the primary optic centres in the pregeminum and pregeniculum, if the lesion is behind these centres, the reaction does not occur. If the lesion is in front of the primary centres, the inaction occurs.

Colour Changes.—Occasionally subnormal colour perception exists with hemianopsia or with sector defects of the eye. Concentric restriction in the field of vision and reversal of the order of colour field may be present and may be tested for with colour disks in the slide of the perimeter. Abnormalities of the visual fields may occur in functional disease; thus, in hysteria we may have complete reversal of the colour field with a constriction of the field of vision. The condition is likely due to defect of perception in this disease, as described by De Schweinitz and J. K. Mitchell. The test for colour is made by means of coloured yarns.

Pupillary Symptoms.—We note under this if the pupils are dilated, contracted, or irregular; also seek for abrupt and frequent changes in the pupil and also for spasms or paralysis of accommodation, etc.

Argyll-Robertson pupil is one in which there is wanting response to light, the power of accommodation remaining.

A phenomenon called *hippus* is produced when the hand covering the eye, directed towards the light, is removed, the pupil at
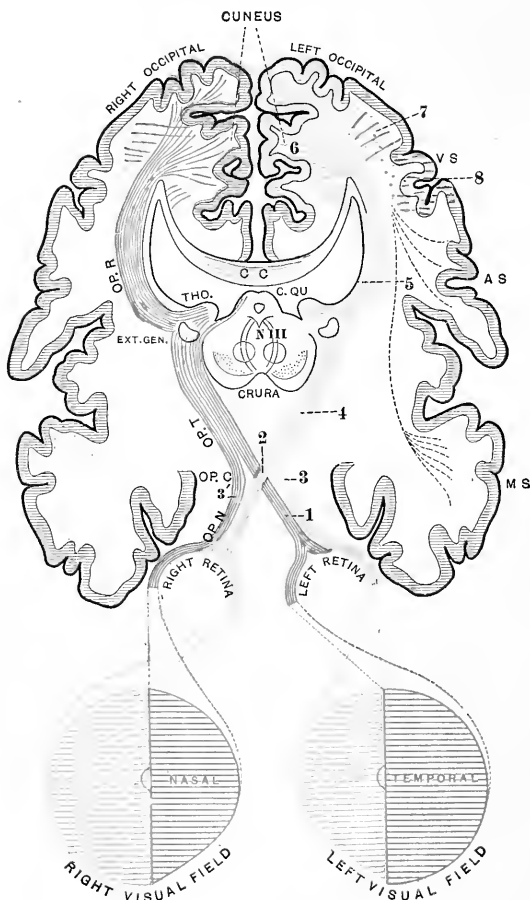
FIG. 19.—DIAGRAM OF VISUAL PATHS. (From Vialet, modified.)

OP. N., Optic nerve.   OP. C., Optic chiasm.   OP. T., Optic tract.   OP. R., Optic radiations.   GEN., Geniculate body.   THO., Optic thalamus.   C. QU., Corpora quadrigemina.   C. C., Corpus callosum.   V. S., Visual speech centre.   A. S., Auditory speech centre.   M. S., Motor speech centre.   A lesion at 1 causes blindness of that eye; at 2, bi-temporal hemianopia; at 3, nasal hemianopia.   Symmetrical lesions at 3 and 3 would cause bi-nasal hemianopia; at 4, hemianopia of both eyes, with hemianopic pupillary inaction; at 5 and 6, hemianopia of both eyes, pupillary reflexes normal; at 7, amblyopia, especially of opposite eye ; at 8, on left side, word-blindness.

first contracts, and then slightly dilates, and thus oscillates until it settles to the original size.

During the testing for mobility of the iris the eye must be fixed upon a distant point, if not, the influence of accommodation and convergence will prevent accuracy.

Hearing.—This is tested by means of a watch brought towards the ear, the point at which it is first heard to tick being recorded. The eye should be closed when this is being done. This test shows the extent of air conduction. Bone conduction is tested by using watch or tuning-fork. The patient being deaf, a tuning-fork struck, then placed over the mastoid process is heard better than by aerial conduction in an ear that is diseased in the middle portion. Disease of the labyrinth or nerve will, of course, destroy bone conduction as well as aerial.

Galton's whistle is a piece of brass tubing, with an internal diameter of less than $\frac{1}{10}$ of an inch, into which a plug can be fitted and drawn in or out at will. This is used in determining pitch.

Deafness is of different degrees. We speak of this as a person being *totally* deaf, *partially* deaf, or having *impaired hearing*. The three terms indicate the degree of defect in the order mentioned.

Smell.—In testing the efficacy of the olfactory nerve, and in distinguishing acuteness, we should avoid, first, the use of any irritating substance, and, secondly, select pleasant odours; thus, the odour of violets, musk, camphor, oil of cloves, etc., should be placed upon cotton and applied to one nostril while the other one is held closed at the time. The loss of the sense of smell may be due either to a functional or an organic disease. Of the former, hysteria is an example. *Anosmia* is the name given to that condition where there is loss of the sense of smell. Fracture through the ethmoid may cause organic anosmia. *Hyperosmia* is increased acuity of the sense of smell. *Parosmia* is the perversion of the sense of smell.

Taste.—This is tested for by means of a sweet solution, or of something sour or bitter. In making the test the patient should be asked to protrude the *tongue*, the solution being dropped upon one side of *the tongue*, and the patient again asked to record the sensation before retracting *the tongue* into the mouth, otherwise the sensation will be perverted by impingement of the test solution upon adjacent parts of the mouth. A test should be applied to the

sides, back, tip, and middle of the tongue, according to the part desired to be tested, and is indicated upon each half separately.

The sense of taste can be tested electrically by an instrument invented by Newman consisting of a long stem carrying two wires isolated from each other, at the ends of which are two little balls, forming the poles. The current being applied, the sense of taste, if present, is readily perceived in a sapid and metallic sense of taste. In order to prevent confusion, smell, touch, or taste, the eyes should be shut and the nostrils held in difficult cases. *Ageusia* is absence of taste. *Hypergeusia* is increased sensibility of taste. *Parageusia* is perversion of the sense of taste.

Reflexes.—Since these are most important they will be gone over again from a different viewpoint. The reflexes are divided into cutaneous, deep, and visceral, as noted in a previous section.

Exaggerated responses of skin reflexes will be produced if the special sense organ is in an irritated or inflamed condition, or if they are cut off from central control, or if cerebral sensory areas are diseased. The lack of response would indicate that the nerve-tracts in the periphery or in the cord are injured, or that the special sense organs are destroyed or impaired. The skin reflexes have been divided in accordance with the areas most conveniently tested, as the *supraorbital*, the *epigastric*, over the epigastric region; *cremasteric*, brought out by irritation over the inner part of the thigh; the *abdominal*, over the sides of the abdomen; the *plantar*, obtained by irritating the sole of the foot. In the trunk and upper extremities we have the *intercostal*, the *interscapular*, the *palmar*, the *bicipital*, and in the head the *conjunctival* and *pharyngeal*. The skin reflexes are very easily exhausted even in very sensitive individuals. Therefore, the test should be made quickly and recorded at once before the loss occurs.

Deep Reflexes.—These deep reflexes are muscle or tendon reflexes. The knee-jerk or patellar reflex is that produced by tapping the ligamentum patella, which by stimulating contraction of the erector femori group of muscles, produce extension of the leg. The jerk is due, first, to the direct stimulation of the muscles, and, secondly, to reflex influences. The elbow-jerk is produced by tapping the triceps tendon, the arm being pendant over some object, as the back of a chair. Extension of the forearm occurs. The biceps-jerk is produced upon striking the biceps tendon of the arm, the member being held in semi-flexion. This produces

flexion of the forearm upon the arm. *Jerks* or reflexes can also
be obtained over some of the muscles of the neck, as well as from

SHOWING THE MECHANISM OF THE DEEP
REFLEXES AND EXAMPLES OF THE LESIONS
WHICH MAY INCREASE OR ABOLISH THEM
AS ILLUSTRATED BY THE KNEE-JERK.
   DOTTED CIRCLES = LESIONS ABOLISHING
THE REFLEXES.
   BLACK CIRCLES=LESIONS EXAGGERATING
THE REFLEXES.
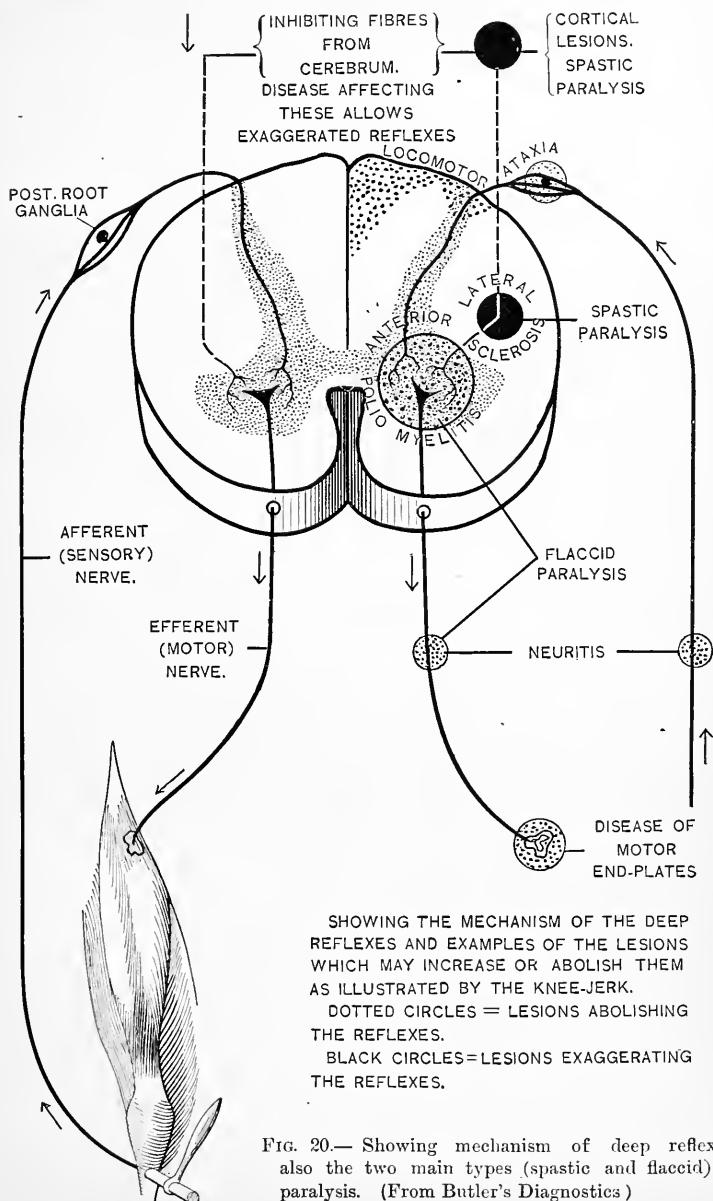
FIG. 20.— Showing mechanism of deep reflexes;
   also the two main types (spastic and flaccid) of
   paralysis. (From Butler's Diagnostics)

the tendons. The reflexes may be *exaggerated, depressed,* or *abolished* in nervous diseases. In making a test the part should be well supported and voluntary effort on part of the patient avoided. The *chin* or jaw-jerk is that produced by tapping the mental process of the inferior maxillary bone, the mouth being held loosely open, when contraction of the temporal muscles occurs to the closure of the mouth. It is diminished in neurasthenia. *Muscle-jerks* are the same as deep reflexes and are also obtained by striking the muscle, when shortening occurs. If disease or an injury severs the nerve supplying the muscle, the tendon reflex is absent, but " humping " from a direct blow over the belly of the muscle may remain for a time.

Ankle clonus is produced by sudden flexion of the ankle, when a rapid clonic movement will follow if the phenomenon be present. *Paradoxical contraction* is the sudden contraction as of the anterior tibial muscles, when the said muscles are suddenly shortened by movement of the extremity. These two reflexes are always abnormal, and indicate excess of irritability of an organic nature.

Bulbo-cavernous reflex consists in sudden contraction of the ischial and bulbo-cavernous muscles caused by mechanical excitation of the glans penis. It is produced by placing the left index finger on the bulbous portion of the penis, and with the right hand striking, with a piece of paper, the dorsal surface of the glans. Onanoff believes that its absence is the sign of an organic lesion. If the sexual function is impaired and the reflex is present, the disease is probably of dynamic origin, and favourable prognosis can be given. This reflex is also styled virile reflex by Hughes of St. Louis.

The *anal* reflex has its centre in the third sacral segment. It is found increased in neurasthenia and in myelitis high up, but is diminished in sacral neuritis and posterior scleroses.

Visceral Reflexes.—*Eye.*—The *light reflex* is produced by throwing a ray of light into the eye, when the pupil contracts, to dilate again when the light is removed. The *accommodation reflex* is brought about by causing the patient to look at a near and a far object. In the former case the pupil contracts; in the latter it dilates. One eye should be covered in examining for the reflex of the pupil. When the light reflex is lost and accommodation remains, we have what is known as the Argyll-Robertson pupil.

Reflexes of the Bladder, Rectum, and Sexual Apparatus.— Urination and defecation are reflex acts under control of higher centres. If the inhibitory influence is removed and voluntary control over the sphincters is lost, urine and fæces are expelled as soon as bladder and rectum are full. Disturbance of function of the pyramidal tracts will bring about this condition. The bladder walls may become weak, and if the *compressor urethræ* remains intact the bladder becomes distended, and we have *incontinence of overflow*. If *any* of the components of the reflex are involved, incontinence will occur. If the motor part is diseased, the sphincters are relaxed. In case of the rectum, if the rectal sphincter is relaxed it is due to (as can be shown by digital examination here) disease of the motor part of the arc. If the *sensory* part of the arc is diseased, the patient is unaware that the bladder or rectum is full. Damage to arc of *genital functions* may bring about loss of sexual power and desire.

The Surface Temperature.—It is desirable to note the local temperature in many diseases, in some of which this symptom is of special value, as in cerebral hæmorrhage, infantile palsy, and in the various vaso-motor and trophic disturbances. In cerebral diseases we may find an increase or a diminution over some portion of the scalp.

In *erythromelalgia* the surface temperature is increased, especially when the affected limb is pendant. This can be detected by a thermometer, which was first invented and constructed by Seguin.

The Mattson's surface thermometer is an instrument consisting of a coil containing the mercury and extending up a tube at right angles to the coil. It is the best instrument that we have used.

*GENERAL THERAPEUTICS AND PREVENTION OF NERV-
OUS DISEASE AND THE ACQUIREMENT OF NERVOUS
HEALTH — HYDROTHERAPEUTICS — MASSAGE — ELEC-
TRICITY — HYPNOTISM — CARE AND TREATMENT IN
CONVALESCENCE FROM FUNCTIONAL NERVOUS DIS-
EASE—CLIMATOLOGY OF NERVOUS DISEASE*

TREATMENT AND PREVENTION OF NERVOUS DIS-EASE AND THE ACQUIREMENT OF NERVOUS HEALTH

In the treatment of nervous disease the physician should at-tempt to relieve distressing symptoms, or better, to secure an out-and-out cure, or to prevent return—prophylaxis being always the ideal, but which it is difficult to inculcate into the minds of or enforce upon people who have not once gone through suffering.

General measures to be instituted in the care of nervous dis-ease are, first hygiene, then exercise, rest, diet, climate, hydro-therapy, massage, electricity, external and internal applications and measures, surgical procedures, and finally, the judicious use of drugs and of other remedial measures.

Hygiene.—To maintain normal nervous systems in the present social life led by Americans would require extreme alterations in method of living of the majority, though gradually the cultured educated classes are beginning to appreciate this, and are adopt-ing more common-sense methods of living, which wise scientific doctors have been advising for so long. Methods here detailed are particularly applicable to the neuropathic individual, but it must be remembered that the nervous predisposition does not start *de novo*, and hence the suggestions to be here made can apply in proper measure to all active people.

Thus, persons of neuropathic stock should not intermarry. Consanguinity should be avoided ; nor should intermarriage take place betweeen families of highly nervous temperaments. Chil-dren should live much in the open air to develop themselves by

natural methods of physical exercise, such as walking, boating, horseback-riding. The overdoing of many so-called medical gymnastic exercises should be prevented. People should be taught to eat slowly, masticate and insalivate thoroughly, and to live on a wholesome *mixed* diet of fats, carbohydrates, and proteids. Self-control and obedience should be inculcated into children from an early age. This particularly prevents the development of hysteria, that disorder of the emotions. Systematic study and work is essential; never overwork in growing youth. All reflex causes, such as by eye-strain, through poor light, or errors in refraction, should be looked to. Ventilation, if poor, will have a baneful influence through preventing proper oxidation and growth and nutrition of nerve and other tissues in the economy. The occupation should be carefully selected for the young; *over*education is to be avoided, since it brings about stress upon the growing brain and its highest function, the mind. Weak children should indeed. be especially educated in accordance with their physical development and nervous capacity. Precocious children are only more apt to develop breakdowns because they *do* overwork. The queer or eccentric child is the one, however, that must be well guarded if a nervous wreck is not to develop at adolescence. Mental overwork in them should largely be substituted by physical exercise. Even this can easily be overdone. Many people of this stamp would make excellent artisans or mechanics, or even labourers, and enhance the value of the race by such manual labour rather than by being advised or set to do the work of the mind such as is compassed in the professions. " Moderation in all things " is an old adage that amply applies to prophylaxis of nervous disease. This applies to sexual indulgence in adults in particular. Exercise is a very valuable prophylactic measure for them. Luetic infection is the one prominent single misfortune that can happen to an individual; for even if treated to the full, there still lurks remote toxines within the blood which at the degenerative period of life cause neurasthenia, hysteria, or grave organic or suborganic disease, which seldom can be entirely relieved. The overuse of tea, coffee, condiments, tobacco, or alcohol must be remembered as causes of various functional and organic diseases, such as optic atrophy or chronic neuritis. Mental and physical trauma ; poisons, as lead or mercury; or the infective fevers, are all causes of various nervous disorders. Alcoholic abuse, it should be repeated, stands

next to syphilis as a cause of a legion of diseases of the nervous system. At least eight hours of sleep should be had in twenty-four hours by every healthy man.

Diet.—As indicated above, nitrogenous food should be given in abundance to the nervous, since there is waste in them of nuclein and other proteid substances from the nerve-cells. Fats are next in importance; carbohydrates least, and they also frequently interfere with digestion, since it requires more caloric, and therefore nervous energy to oxidize this class of foodstuffs. Water should be drunk freely between meals, and even a glass of water at the meal is a desideratum, since certain salts of food need the chemical action of water to convert them into soluble chemical substances. The obese should not eat or drink as freely as a spare person. If muscular exercise is indulged, then the total amount of food should be increased. If the patient is constipated, green vegetables, farinaceous foods, or an orange before breakfast are valuable remedial measures in dietary. The best foods are meats—beefsteak and fowl; also eggs, fish, milk, buttermilk, and cocoa; stale bread, to which can be added plenty of butter. Metal workers should have plain drinks and alkaline waters. It must be remembered that adults cannot take so copiously of milk as children. The use of alcohol is baneful for the nervous body. Some claim it may increase capacity for work, but I am perfectly sure this is not the case. In disease with temperature it is, of course, of value.

" Generally nervous " still applies to a class of irritable persons annoyed by trifles, yet not truly neurasthenic nor hysterical. In all three of the latter states, however, the patients can and should take large quantities of proteid food, to which should be added the heat-giving foodstuff fats as much as can be easily digested, since these patients always complain of being cold or chilly at the slightest exposure. Some nervous persons cannot take sweets without producing headache, rheumatic pains, or disorders of digestion, due to the fermentation produced and acid condition of the blood (uricacidæmia) following. In such people the diet should exclude carbohydrates and include meats, fish, oils, as cod-liver or olive; also cream, milk, oysters, and even pork. Spinach is an excellent food for the neurotic, since it is laxative and contains a goodly proportion of iron. The white of an egg added to beef-tea, to which can be placed in addition some form of peptonoids,

is very nutritious. Besides the stale bread indicated, in which fermentation is complete and the individual elements better dispersed, the nervous person can use the special forms of breads in which the starch has largely been removed. These give the gastric content a release from overtaxation in digestion of the carbohydrates. Practically, however, the patient cannot take these breads for any length of time, since they are unpalatable.

If a *rigid diet* is desirable make it as follows: Milk, oysters, butter, eggs, raw or soft, cocoa, graham or gluten bread, beef, fowl, mutton, lamb, or fish. Among vegetables spinach, lettuce, string-beans, Brussels sprouts, or stewed fruits are the best. The neurotic person, from the subjective craving of nature (boulimia), is liable to eat too much. Hence this should be guarded against, since overloading the stomach in them may cause gastrectasia, an added mechanical cause for indigestion. *Tea, coffee, and alcohol are baneful in effect upon the frankly nervous person.* Overuse of the alkaline mineral waters should be avoided, since disturbance of the normal acid secretion of the stomach will be brought about and add to distress. Three to four pints of plain water should be drunk *per diem.* Dryness of or desiccated nervous systems cause instability of function.

Exercise.—The value of exercise, if taken out of doors, as in rowing or horseback-riding, where the mind cannot run in special grooves, is a certain preventive of nervousness beyond calculation. The exercise should not be violent in brain-workers—i. e., they should not go into severe athletic training. Even the majority of youth or of adults cannot do well both physical training and mental work of the highest sort. Nervous persons need exercise associated with interest to the mind; hence the individuality of the pleasure of one person as compared to another must be borne in mind, and not too strict rules laid down by the physician. Gymnastic exercises indoors, as a rule, do little good from the fact of the monotony of the procedure entailed. The mind must be properly exhilarated with it all. Walking is a valuable form of exercise, since it is moderate, and movements of the arms bring into play the muscles of the chest. Horseback-riding is the very best exercise the nervous patient can take, since it carries the patient out into the fresh air, expands the chest, gives free use of the arms, and does not require the extremes of exertion as with other forms of exercise. Then the spinal cord is not drawn upon so

heavily in expending the little reserve of nervous energy the neu-
rotic holds claim to as does walking, bicycling, golfing, and the
like. Exercise should be cut down much after the degenerative
period of age has set in (i. e., after forty) if a person wishes to
live a long life.

HYDROTHERAPEUTICS IN NERVOUS DISEASES

This important adjunct to neurological care of the many cases
coming under our observation is important for the physician and
nurse to know well. The proper external use of water aids much
the general treatment of nervous disease. The effect of water be-
sides the hygienic place, and often the favourable mental influence
upon the patient, as in cases where neurasthenic and hypochondri-
acal phobias exist, is mainly due to reflex action upon the nervous
system. The " reflex " arcs so patent in the symptomatology of
organic diseases of the central and peripheral nervous systems we
are apt to let pass by when we are caring for the so-called func-
tional diseases; whereas in them can be found the reflex guide-posts
to the treatment of the case that may be cured. In organic dis-
ease, it must be re-enforced, we have the grosser expressions of
what scientific clinical medicine behooves us to·search for in the
very incipiency. And it is here in place to say that the scientific
physician will ever be the leader in progress, albeit medicine, for
the very reason that we deal with life, can never be a true science.
Much of the treatment at Carlsbad is scientific; a great deal of the
good results are from the engendered mental buoyancy which we
must recognise. With these well-balanced therapeutic ideas kept
perfectly clear in mind, the physician of largest attainments will
use bathing freely more and more conscientiously until he learns
the true value of the bath in its physical effects on the body, sick
or well.

Turkish Bathing.—The origin of Turkish bathing, so peculiar-
ly associated with the religious rites of the oriental people, is quite
as much *in nubibus* as is the origin of language. The effects of
dry heat are finally to stimulate hyperæmia of the surface of the
body, to cause hypercaloria and hyperhydrosis. As the heart is
stimulated by increase of temperature, that organ must be very
competent before a patient is relegated to this initiative step of
the Turkish bath. Indeed the heart should be toned up by digi-
talin, strychnine, or strophanthus when there is the suspicion of

cardiac weakness. A patient with pronounced valvular disease should be prohibited from Turkish bathing. Persons suffering from obesity or interstitial nephritis may be greatly alleviated of distressing subjective phenomena of vertigo, headache, and depression by judicious use of Turkish baths. This beneficial effect must be due to elimination of toxines always retained in such cases.

In anhydrosis, as in myxœdema, or in cases of atrophic dry skin I have so often seen in women at the climacteric suffering from general nervousness, Turkish bathing does a great good. In cases of *neurasthenia terminalis* I have seen much improvement through this form of bathing.

Hystero-neurasthenia in persons of gouty diathesis, on the other hand, are not benefited at all by the Turkish or Russian baths.

Details of the Turkish Bath.—Like the massage we use in America, which is the resultant of selection from the best methods, the Turkish bath is *modified* as we use it for best therapeutic results. The patient should have at least two hours to give to the bath. This will allow amply for the one and three-quarter hours which should practically be the time consumed in the procedure. The patient is at first sent to the dry hot-air room, where he sits in a reclining chair, keeping perfectly quiet and relaxed. If he reacts favourably, beads of sweat will soon begin to come to the surface of the body, which is always a favourable aspect. Should the patient complain of sense of faintness, the application of cold cloths to the head by the attendant is directed; also the drinking of cool water will do much to relieve this symptom and to hasten hydrosis. As before stated, the patient should be allowed to remain some fifteen or twenty minutes. The temperature of the room is kept up by means of hot water to the average of 153° F. In cases of obesity, the patient may next be taken to the hotter room adjacent, the temperature being as high as 175° F. The attendant must constantly observe the case taking an initial bath in order to avoid any ill effects of *idiosyncrasy*. The patient is next taken to the *rubbing room* in moderate temperature and placed upon a marble slab on his back, with his head and heels protected by cushions or sponges.

He is then given a thorough surface massage by the attendant, the general movement being from periphery to the trunk. With

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this there is a series of slapping motions with the palms of the hands, which greatly stimulates the circulation. The back is then treated in the same fashion after the patient is placed in the prone position. The water from a hose at about the temperature of 105° F. is frequently deluged upon the subject with some considerable force during the manipulations. He is finally " soaped over " thoroughly and again " washed down " when he is prepared to enter the steam-bath. Then the " shower " is had, or he takes the " plunge " in the bath, at the discretion of the physician.

In the *steam-bath* lies the *modification and blending* of the Turkish and Russian baths used to-day. The patient enters the door and quickly proceeds to the opposite side of the room, where he sits on the marble slab quietly for about five minutes; this is likely to be the most discomforting part of the entire procedure, and the patient should be carefully watched by the attendant in the first entrance, for there occasionally comes a sense of suffocation, which is more a mental symptom than that it is due to lack of oxygenation of the blood. Otherwise at this stage there is a comfortable feeling, the sweat seeming to pour from the individual, which, in reality, is not so, but is due to condensation of vapour upon the body. The temperature of the body is actually reduced by this part of the bath, and this is the only reason why the vapour part of the bath should be so short.

The *shower-bath* should be substituted in all cases of first treatment in order to gain the confidence of the patient in the rather shocking changes. The shower-bath is administered by a series of sprays tempered to the patient physically, and it is essential that the water should pour in these fine streamlets upon the head as well as the rest of the body, else there may be a tendency to congestion of the brain and its membranes. Temperature should range from 65° to 85° F. If the plunge is taken, the patient should immediately immerse himself in the pool. A good way to do this is by diving, if he is familiar with the water. He should remain in the water three or four minutes, the average temperature being 65° F. The patient is next dried and rubbed in the standing posture by the attendant. He is then wrapped in a sheet and blanket and placed upon a couch or bed, to lie quietly for at least half an hour, sleep usually following.

HYDROTHERAPEUTICS—*Continued*

Water may be used either as a *tonic* or as a *sedative.*

As a *tonic* we employ cold plunges, shower-baths, various forms of douches, as Charcot's and the Scottish cold sitz-bath, salt baths, either sea or artificial, and short cold packs. These all have a stimulating and tonic effect. In giving them, especially to weak people, it is best to begin with warm water and gradually lower the temperature. Showers and douches are the most stimulating. A reaction should always be obtained by vigorous rubbing afterward.

For the *cold plunge*, the patient immerses himself in the bath of water at a temperature of 60° to 70° F., and at once emerges. He should then be rubbed vigorously.

The *shower-* or *rain-bath* consists in allowing water to fall on the body from a height for one or two minutes while the feet are in warm water. Friction of the body should be kept up during this process.

A *Charcot douche* is given by directing a solid stream of water with force upon the back of the patient.

By the *Scottish douche* we mean alternating a cold douche with a warm or hot one.

Cold packs are given by wringing a sheet out in cold water, wrapping about the patient for a few moments, when it is removed and the patient put to bed and rubbed.

An *artificial salt bath* may be made by putting 25 pounds of salt (NaCl) in 30 gallons of water (2 per cent). It may be warm or cold.

To obtain *sedative effects* we may use the lukewarm bath, wet pack, hot sitz-bath, hot compresses, and drip-sheet.

The *lukewarm bath* is given at a temperature of 95° to 98° F. for ten to twenty minutes.

To give a *wet pack*, spread a large, thick blanket upon the bed; upon this is laid a sheet wrung out in water at a temperature of 40° to 60° F. The nude patient lies upon this and the sheet is wrapped smoothly about him, not including the head and feet. The sheet must be carried between the legs and brought evenly in contact with the body. The blanket is then folded over him, with others added if desired. Hot-water bottles may be placed at the feet and cold compresses to the head. The patient lies in this for half an hour, and is then thoroughly rubbed.

The Drip-Sheet.—Have a basin of water at 65° F. Put in the basin a sheet. The patient stands in comfortably hot water. Have ready a large soft towel and iced water. Wring out the towel in this and wrap it around the head and back of the neck. Standing in front of the patient, the sheet is seized by the two corners and thrown about the patient, who holds it at the neck. It is then smoothed out over the body; next loosened, dropped, and the patient is instructed to lie down on a blanket, which is wrapped about him. Dry thoroughly with coarse towels, wrap in a dry blanket for a time, then put to bed. The water should be gradually cooled day by day until it is 55° F.

The bath, pack, and drip-sheet are valuable remedies for insomnia.

A *hot sitz-bath* consists of water at a temperature of 100° to 125° F. In this the patient sits from twenty to thirty minutes.

Hot compresses are often used for the relief of local pains and congestions.

MASSAGE

Massage has been used as a therapeutic agent in more or less crude fashion for centuries.

The wrestling matches of the Greeks left many a lame back or joint which their rubbers dissipated by manipulations. We all know, too, the uses of rubbing in alleviating the sprains and bruises of our army of college athletes.

By greater refinement in the *modus operandi* of massage, and by very careful analysis of the effects to be obtained by each individual movement, this useful adjunct has been in recent years placed on a much higher plane in the therapeutic arts; indeed, in some instances, in expert hands it has almost amounted to a scientific basis.

Schools of massage have been established where lectures are given in the larger cities of this country, also thorough practical work is obtained during several months' course required to become experienced in rubbing. At the end of this time an examination is had, when a certificate is given entitling the holder to recognition by the physician. Then a course in electricity is added to complete the training of one who chooses the vocation of masseur or masseuse. The two (massage and electricity) go so completely hand in hand in the treatment of the nervous mal-

adies, where they are chiefly of value, that it is necessary to be able to administer either one or both under the direction of the medical man.

Notably in American schools very good training is given, but also abroad, especially in Germany and Sweden, in which latter country the series of movements bearing the name of Swedish are perhaps best known in a general way. In America all these very intricate movements have been somewhat modified; and while the general tendency is towards definite ends, yet the extremely rigid mechanical side of massage is largely done away with, and we have a more perfect system, much better suited to the delicate human organism.

The Five Sets of Movements of Massage.—1. Effleurage is the gentle surface stroking of the part which quietly starts the circulation before the more vigorous.

2. Friction is then begun. This latter consists in a firmer and deeper pressure-rub than the preceding movement.

3. Pétrissage, which is a very deep kneading of the part and completes what has been begun by 1 and 2. It is essential in pétrissage to hold firmly to the skin and to make this rub the subcutaneous tissues, while the last in turn presses the muscles, and so on until the soft parts are so manipulated, squeezed against the bones of the patient, that a veritable pushing on all of the liquids (blood and lymph) takes place; also, indeed, of some of the semi-solids, which are urged to disintegration and their ultimate particles swept on in the hurried circulation, to be oxidized or converted into energy, or to be excreted, instead of remaining in the body as sources of irritation in the form of what we call leucomaines.

4. After this thorough application a movement, not so very essential, is yet quite often used as a final stimulator of the circulation where it is extremely sluggish. It consists of *tapotement* or tapping in a rapid vibratory manner with the balls of the fingers of one or both hands held gently closed, similar to the position of holding a pen.

5. Next and last comes effleurage again, by which the harder rubbing now reached shades off to less vigorous frictions. These are soothing to the part and to the patient generally, while they also equalize the blood circulating in the superficial tissues. Then the member is immediately covered with a light woollen garment,

and when the entire body is so manipulated the patient rests for an hour.

Modified Swedish movements, *passive* then *active* (*without* and *with* muscular resistance of the patient), are often instituted after a course of general massage of some weeks' duration. We are now speaking of general rubbing or applying the art to the entire body, as in cases of neurasthenia, where partial or the complete " rest cure " is being systematically carried out.

How to " Rub " a Patient.—First the subject is required to thoroughly relax all muscles; then he is placed on the right side, and the manipulations are begun on the left foot, toes, ankle, thigh, hip, and buttock in turn, using all the five movements described over each part, varying the time and duration of each in proportion to the size, rigidity, and amount of muscle or other soft tissues present. The patient is then asked to turn towards the opposite side, and the right foot, leg, thigh, and buttock are rubbed in the same thorough fashion.

Gradually one set of movements dovetail into the other, and the patient's extremity becomes almost a part of the rubber, as does the dough of the housewife; and yet you note that by the time the limb is finished every nook and cranny has been gone over, leaving a beautiful glow of the surface in evidence of the vigour set up in the circulation of the blood.

Next the left fingers, forearm, arm, and shoulder are first given the long sweeping effleurage, followed again by the rest of the detailed movements described; but the eye has to follow quickly to appreciate the rapid changes, so much a part of the manipulator does the patient become. Four or five minutes will suffice, and then the right arm is taken up. After this we request the patient to lie on the abdomen with a pillow placed under for better resistance. The long series of strokings down the vertebral gutters made alternately with the fingers of each hand spread on either side of the spinous processes. Then comes a series of circular frictions down each recti group with one hand, then a spreading movement from the spines outward with the balls of both thumbs; next a firmer pétrissage with both hands, followed by that motion with the palms of both hands alternately run down either side of the spinal column, giving a delightful sensation, while the final effleurage finishes and the patient is requested to turn on the broad of the back, the pillow being removed.

The chest movements consist principally of two : a firm strok-
ing, following the ribs from the sternum out and down, and of
firmer pétrissage, in which the balls of both thumbs play an im-
portant rôle, and can be so dextrously done as to be not at all
painful, which occasionally happens with the beginner.

The abdomen is rubbed as follows (this is very important and
difficult to acquire) : the thighs are flexed on the abdomen and
legs on the thighs in order to relax the anterior abdominal walls.
The effleurage is simple enough, but it requires tact to knead thor-
oughly without tickling. Begin this over the small intestine and
work with both hands in a " spanning "-like manner. Then start
at the caput coli and work along the ascending transverse and
descending coli, one hand following the other in rotary motions.
Again effleurage follows, and, as pointed out above, if constipation
is an indication to be met, that series of rapid vibrations described
will aid much towards the cure. As a rule, the face, head, and neck
are not rubbed in ordinary work. Where especially requested, it is
given by a series of strokings and kneading movements in the
direction of the venous circulation. It should be stated that in
all movements the greater pressure should be from the periphery
to the heart—i. e., centripetal in character. The patient is now
allowed to have a quiet sleep.

The **salient points of massage** are for the masseur to keep his
hands soft, clean, dry, and warm.

A general rub should last from one half to one hour, preferably
about 10 A. M., or at bedtime. If electricity is used, give it at the
opposite hour. The patient should rest at least three-quarters of
an hour afterward. In orthopædic work a maxim obtains—viz.,
" to tighten a loose joint rub easy, to loosen a firmly fixed joint
rub hard and deep." The relation of gymnastics to massage
we have only time to hint at ; suffice it to say they are closely
allied, and the former are perhaps of greatest value in lateral
curvature of the spine and in aiding chest expansion in lung dis-
ease. These regulated movements should also be under observation
of the physician.

It has been proved that the renewal of the epithelium of the
alimentary tract is made more active under massage, and that it
stimulates peristalsis. Blood-pressure rises after massage, and
Dr. J. K. Mitchell's investigation [1] shows that the blood elements

are put into more vigorous circulation during and after this treatment. Massage is of great value for the surgeon in getting rid of exudates about old fractures.

As to the matter of technique—of course some become more skilled than others, but any person using tact and practice will learn to rub well. The therapeutic results obtained from massage we have not time to consider further, except to mention the very frequent relief of neuralgia by its careful use.

Contra-indication to Massage.—It should not be administered for an hour after a meal. Fever is a contra-indication to massage. Enlarged and thrombosed veins should not be rubbed, else a great risk of setting up a local active inflammation ensues, or that particles (emboli) may be carried on to some important organ, as the brain, causing paralysis or other serious results. Tumours, especially malignant ones, should not be manipulated for fear of exciting them to more rapid growth and metastasis. If massage is extremely irritating after a fair trial it should be discontinued. There will occasionally be found one in fifty who is made worse by rubbing. In these persons we must resort to gymnastics alone or to electricity. The pregnant woman should not be rubbed about the abdomen; and it is advisable to abstain from abdominal massage during menstruation, also in any case of acute pelvic disease. Bimanual pelvic massage may be of value in chronic cases with fixation of the uterus from old adhesion.[1]

MUSCULAR MOVEMENTS

Regular *muscular movements,* according to a fixed schedule, as first used by Weir Mitchell, but since elaborated by Fraenkel, are of value in the treatment of muscular inco-ordination. The exercises should be performed with care and precision twice daily.

The following schedule, based upon that of Fraenkel [2] and Hirshberg, is recommended by Dana :

Exercises for the Hands and Feet.—1. Sit in front of a table ; place the hand upon it, then elevate each finger as far as possible. Then raising the hand slightly, extend and then flex each finger

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[1] For more detailed and exhaustive study the works of William Murrel, M. D., F. R. C. B., on Masso-therapeutics, 1890, and of Emil Kleen, M. D., Handbook of Massage, are recommended.

[2] The Treatment of Tabetic Ataxia, etc., 1902.

and thumb as far as possible. Do this with the right, then with the left. Repeat once.

2. With the hand extended on the table, abduct the thumb and then each finger separately as far as possible. Repeat three times.

3. Touch with the end of the thumb each finger-tip separately and exactly. Then touch the middle of each phalanx of each of the four fingers with the tip of the thumb. Repeat three times.

4. Place the hand in the position of piano-playing and elevate the thumb and fingers in succession, bringing them down again, as in striking the keys of the piano. Do this twenty times with the right hand, and the same with the left.

5. Sit at a table with a large sheet of paper and pencil. Make four dots in the four corners of the paper and one in the centre. Draw lines from corner dots to centre dot with the right hand; same with the left.

6. Draw another set of lines parallel to the first with the right hand; same with the left.

7. Throw ten pennies upon the paper. Pick them up and place them in a single pile with the right hand, then with the left. Repeat twice.

8. Spread the pennies about on the table. Touch each one slowly and exactly with the forefinger of the right hand, then with the forefinger of the left.

9. Place an ordinary solitaire board on the table, with the marbles in the groove around the holes. Put the marbles in their places with the right hand; same with the left hand. Patient may, with advantage, practise the game for the purpose of steadying his hands.

10. Take an ordinary fox-and-geese board with holes and pegs, and beginning at one corner place the pegs in the holes, one after the other, using first the right hand and then the left.

Exercises for the Body and Lower Limbs.—1. Sit in a chair. Rise slowly to the erect position without help from cane or arms of chair. Sit down slowly in the same way. Repeat once.

2. Stand with cane, feet together; advance the left foot and return it; same with the right. Repeat three times.

3. Walk ten steps with cane, slowly. Walk backward five steps with cane, slowly.

4. Stand without cane, feet a little spread out, hands on hips. In this position flex the knees, and stoop slowly down as far as possible; rise slowly. Repeat twice.

5. Stand erect, carry left foot behind, and bring it back to its place; the same with the right. Repeat three times.

6. Walk twenty steps, as in Exercise No. 3; then walk backward five steps.

7. Repeat No. 2 without cane.

8. Stand without cane, heels together, hands on hips. Stand in this way until you can count twenty. Increase the duration each day by five, until you can stand in this way while one hundred is being counted.

9. Stand without cane, feet spread apart; raise the arms up from the sides until they meet above the head. Repeat this three times. With the arms raised above the head, carry them forward and downward, bending with the body until the tips of the fingers come as near as they can be safely carried.

10. Stand without cane, feet spread apart, hands on hips; flex the trunk forward, then to the left, then backward, then to the right, making a circle with the head. Repeat three times.

11. Do No. 9 with heels together.

12. Do No. 10 with heels together.

13. Walk along a fixed line, such as a seam on the carpet, with cane, placing the feet carefully on the line each time. Walk a distance of at least fifteen feet. Repeat twice.

14. Do the same without cane.

15. Stand erect with cane; describe a circle on the floor with the toe of the right foot; same with the left. Repeat twice. Between the fifth and sixth exercise the patient should rest for a few moments.

ELECTRICITY

Physics.—The laws which govern the electrical current are similar to those governing the flow of water. If two vessels one above the other and connected by a tubing, and water is poured into the higher vessel, it will tend to run to the lower vessel; the water will thus generate force or capacity for work which is called *potential*. The fluid in the lower vessel would also tend to reach a low level, but the force would not be so great as that of the higher vessel; at sea-level would be zero potential. The difference

in force exerted between these two bodies of water would be their difference in potential.

By means of certain agencies (chemical change, friction) electricity is separated into positive and negative electricity, the positive being of higher potential than the negative, the positive tending to flow towards the negative and thus cause an electric current. The zero point of an electrical current is the earth.

Volt.—The force which starts the column of water flowing is gravity; that force which causes an electric current to flow is called electro-motor force (E. M. F.). The unit of measurement of this is called the *volt*.

Ohm.—Some substances are much more pervious to the passage of water than others. This is so with electricity. *Conductors* permit the electric current to flow easily. Metals, plumbago, dilute acids, saline solutions, water, and living animals are good conductors in the order mentioned. *Non*-conductors or *insulators* do not permit free passage of electricity—such as rubber, glass, silk, resins, and dry air.

Just as water encounters resistance as it flows through a pipe, so does electricity meet resistance as it passes along a conductor; and this resistance will depend upon the length, composition, and area of cross-section of the conductor. Thus a current passing a *short* distance through a good conductor of large area of cross-section will meet with less resistance than a current when passing through a poor conductor with a small cross-section area, or a long distance. The unit of resistance is called an *ohm*.

Ampère.—The current strength (C) is dependent upon Ohm's law, which is that the current strength is equal to the electro-motor force divided by the resistance $(C = \dfrac{E.M.F.}{R.})$. The current strength is measured in ampères. In medicine we use $\frac{1}{1000}$ part of an ampère, called a milliampère. When electricity is confined, as by means of insulators, it is said to be *static*.

Physiology.—Electricity acts in various ways upon living tissues. It possesses the power of *cataphoresis*—i. e., it may carry solutions through the tissues in the direction of the current. By this means cocaine for anæsthesia and various other drugs, as mercury, may be carried into the tissues. Electricity may also possess the power of *electrolysis* or chemical decomposition of tissues at the electrodes. By this destruction of nævi, or small tumours,

may be brought about. Electricity also causes a modification of
nerve-excitability known as *electrotonus*. In the vicinity of the
anode the excitability is lessened—anelectrotonus; while at and
in the vicinity of the cathode the excitability is increased—cath-
electrotonus. When anelectrotonus is made to suddenly dis-
appear by the breaking of the current at the anode, cathodal
increase occurs, and the nerve is thrown into a condition of cath-
electrotonus.

Electric currents also cause *muscular contraction,* and this is
produced by a sudden increase or decrease of electrical excitement
in the muscle or the nerve supplying it. If this is done but grad-
ually no contraction results. The most powerful contraction is
therefore caused by the *voltaic alterative*. It is produced by sud-
denly reversing the current direction, so that a nerve or muscle
that was in a condition of anelectrotonus is thrown into a condi-
tion of cathelectrotonus, or from a state of — excitability to
one of + positive, and *vice versa*. By simple closure and open-
ing of the circuit the increase is only from 0 to + excitability
and 0 to — excitability, which, of course, is not so great a vari-
ation.

The *galvanic* current produces all of these properties in more
marked degree than does the *faradic* or *static*. In the latter two
forms the influence of the current, due to constant interruptions, is
so brief in duration that they are practically only used to cause
muscular contraction and relieve pain respectively.

A degenerating muscle loses its power of response to static elec-
tricity first, next to faradic, then to simple *opening* and *closing*
of the galvanic current, and finally to the *voltaic alterative*.

Diagnosis and Prognosis.—In many diseases of the nervous
system, particularly cord diseases, the muscles and nerves, when
submitted to electrical stimulation, act differently to normal mus-
cles and nerves. This *electrical irritability* may differ in two ways,
quantitatively and *qualitatively*.

Change in quantity implies a diminution or decrease in irrita-
bility, while change in quality that there is change in the *character
of the contraction*.

Quantitative Change.—*Simple increased irritability,* or that in
which the muscle or nerve responds to a weaker current than nor-
mal, is met with most frequently in tetany. *Decreased irritability,*
or that in which a stronger current than normal is required to pro-

duce contractions, occurs in mild cases of neuritis, and occasionally in long-standing central paralyses. And these conditions are generally the same for both currents.

Quantitative Changes—Examination.—To ascertain these: if the paralysis is unilateral, the weakest current will cause contraction on the normal side should be learned; then the strength of the current that will produce the same result on the diseased muscles is determined, and the two compared.

If the disease is bilateral the reaction may be compared with the reaction of a normal person of similar physique, or judged by experience as to the strength of current usually required in the particular muscles tested.

Qualitative changes consist of *reaction of degeneration* (DeR) and of the *myotonic muscular reaction,* the former being found in certain peripheral palsies, and therefore, when present, always indicates a lesion situated in the peripheral motor neurone—i. e., motor cell of medulla or cord—or in the axones forming the motor nerves that come from this motor neurone; while the latter (*myotonic muscular reaction*) is a symptom of the clinical disease myotonia congenita. (See Thomsen's disease.)

Reaction of degeneration may be, according to the degree of disease of peripheral motor neurones, partial or complete.

Complete reaction consists of (*a*) rapid loss of the power of the muscle and its supplying nerve to react to the faradic current; (*b*) a brief period of *quantitative* increase followed by a decrease when they are stimulated by the galvanic current; (*c*) the *nodal change*—i. e., instead of the short, jerky contraction caused by the stimulation of the healthy muscle or nerve by the galvanic current, the contraction is slow, wavy, and at times tetanic; and (*d*) the *serial change* when the muscle alone is subjected to galvanic stimulation.

The *partial* or *serial change* depends upon a difference in the way the muscle reacts to different poles of the battery, normally the weakest current that will cause a muscular contraction is when the circuit is closed with the cathode *on the muscle* (cathodal closing contraction CaCLC) ; a stronger current will cause an anodal closing contraction (AnCLC), etc. If we represent anodal opening contraction by AnOC and cathodal opening by CaOC, the *normal formula* will be

$$CaClC > AnClC > AnOC > CaOC$$

The *serial change* in *complete reaction* of *degeneration* (DeR) consists of an increase of the AnClC and AnOC over the CaClC; so that one formula would read AnClC = AnOC CaClC CaOC, or as follows: AnClC > AnOC > CaClC > CaOC.

Partial DeR is more usual than the *complete* as just given, and may consist of nothing but the loss of faradic irritability and the *nodal* change, the *series* remaining normal. The series may be so changed, however, that AnClC = CaClC. In *partial* reactions the nerve usually responds normally.

Examination for the Presence of DeR.—Here we first use the faradic current as when testing for quantitative changes. Then use the large galvanic electrode over the sternum, and the other, a small one, over the muscle. This will show the minimum strength of current necessary to cause CaClC and AnClC. If a milliampèremeter is attached, we can note the number of milliampères required; or if not, by noting that the current which causes CaClC will not cause AnClC, or *vice versa,* or that they are equal.

Another method consists in making the electrode positive or negative alternately, using the same strength of current and noting the difference, if any, in the degree and intensity of contractions. In all of these tests there should be an interval of several seconds permitted between opening and closing the circuit.

The *myotonic reaction* is due to a much increased irritability; mild faradic and galvanic currents produce contractions that are tetanic in character, hollows and ridges in the muscle being frequently produced. In the case of the galvanic current AnClC becomes equal to or greater than CaClC. When the galvanic current is allowed to pass without interruption through a muscle, rhythmical contractions, travelling from negative to positive pole occur.

Electricity may at times help in giving a *prognosis*—e. g., *complete DeR* is only present when extensive damage has been done; the prognosis is more grave than when the reaction only shows partial DeR, particularly when in the clinical signs the anterior horns of the cord show involvement. *Per contra* it is better in nerve-trunk lesion *per se.* Prognosis is best in cases where no serial changes occur. In cases where only feeble AnClC can be elicited, it is usually hopeless.

GENERATION OF ELECTRICITY FOR PRACTICAL THERAPY

Electricity is generated in a cell composed of two opposing elements, one being called *positive,* the other *negative,* both of these being placed in the same solution, a good conducting medium, as, for example, sulphuric acid. Then the current is given off from the battery at the softer metal—for instance, the zinc. A current thus set up passes from the negative to the positive pole within the " cell," so called, thence out a conducting wire to the electrode or pole, designated the positive pole, and from which the current passes when applied to the human body. The pole or electrode from which the current passes to the other conducting wire back to the " cell " is called the negative or active pole of the battery. This apparatus, simply described, is designated a galvanic or chemical instrument, and is most potent for action upon the tissues. A practical method of determining which pole is negative or not is perhaps best made by placing the electrodes in water; the one from which bubbles escape is the negative, active, or electrolytic pole. A good point also for remembering which pole is the more active is to be guided by the paradoxical fact that the " negative pole " is the positive one for action, while the " positive pole " is the negative one in regard to its effect upon human tissues. Galvanic electricity produces profound action upon the human body, and is the one used where there are the so-called degenerations, if electricity has been prescribed for the case.

SOME PRACTICAL INDICATIONS FOR GALVANISM

In treating a case of Bell's palsy do not begin the application earlier than from ten days to two weeks following the attack. Apply the positive pole to the nape of the neck, the negative being applied to the distribution of the peripheral nerve upon the face, from 3 to 6 millimetres being used. So with other local palsies where " reaction of degeneration " has been found, such as in wasting of muscles from infantile palsy, or in brachial and sciatic neurites.

In the tachycardia of Graves's disease, the application of from 4 to 6 millimetres of current for as many minutes will often act favourably in slowing cardiac action, through stimulation of the

vagi. Place the negative pole over the apex and the positive over the carotids by an extension of the positive pole in Y-shape.

The removal of superfluous hair can be accomplished through a galvanic current, as follows; by cathode application to the root of the hair within its follicle: For this purpose, a very fine flexible steel needle securely fastened in a plain insulated holder is passed down along the hair to be removed from its follicle. There should be neither resistance nor pain; if there be, the edge of the follicle has been struck and the attempt to introduce the needle must be repeated. The offending hair is held by means of a small forceps, by which it is extracted later.

The circuit is completed by the patient placing her hand upon the wetted sponge anode. The current must be imperceptible, and within twenty or thirty seconds of its passage froth appears about the needle, and a short while thereafter the hair pops out without traction on the forceps. The patient then removes the hand from the sponge, and another hair is selected for the same treatment. This subject is mentioned in detail since it is a valuable means of helping us in the therapeutics of melancholia in sensitive women due to excessive hirsute growth. I have seen excellent results in the mental disorder where other therapeusis had failed.

The galvanic is also *the* current used for the relief of pain, as in neuralgia; and the positive pole should be applied to the painful part in such cases, the negative pole to be applied to some indifferent distant part of the body. It should not be administered longer than five minutes at one séance. When the current is passing and both poles are applied, it is termed " closed," and when one pole is removed or the circuit broken by the spring attachment placed on the negative electrode, it is termed " open." There are certain definite relations in reaction of the muscles to the current. First, when the current is closed, the negative pole, being placed over the biceps muscle—for example, the positive over the dorsal spine—there is contraction well seen at the moment of closing. When we reverse the pole, the positive being placed over the biceps muscle and the negative over the spine, you will see that at closing of the current by the attached spring or interrupter the contraction of the normal muscle is much less marked at closing.

" Reaction of degeneration " is always made with a galvanic current; never determined in palsies the results of the cerebral disease—i. e., of central origin—but is found in lesions of the

peripheral neurone, as in disease of the anterior horns of gray
matter of the cord, neuritis or other degenerative diseases of nerv-
ous tissues, peripheral to and including the multipolar cells of the
gray matter of cord or medulla, as indicated above.

The order of lessening the contraction of muscles with begin-
ning degeneration of nerves, finally represented in "reaction of
degeneration," is as follows:

$$CaClC > AClC$$
$$AClC < CClC$$
$$AClC > CClC > \quad AOC.$$

The formula $AOC > CaOC$ always obtains, however.

Ruddiness appears at the point of contact of the negative pole
upon the skin, as over the biceps muscle, and if the current is made
strong enough or allowed to pass a few moments, a peculiar "ting-
ling" sensation will arise, and finally an actual "burning,"
which, indeed, can go on to excoriation where the current is al-
lowed to pass of too great strength. You will find for use in the
upper extremities an average of 5 to 10 milliampères is quite
enough, as shown by the attached milliampères through its needle
index.

REGULATIONS OF THE GALVANIC CURRENT

The rheostat governs the amount of electricity passing from
the battery to the body. A convenient movable lever with N. upon
the index, when pushed far to the right or left, always points to
the negative pole, and in larger machines the current is obtained
from a series of galvanic "cells." Therefore we can take the cur-
rent in different wards of a hospital by a transfer cabinet, or "con-
verter." For use in private work, we generally carry a galvanic
battery of 20 to 30 cells. (See illustration.)

AXIOMS

A few axioms and terms used in electro-therapeutics it will
be well to define. The force originating in a current is designated
as the *electro-motor force*. The strength of current used upon the
body is equal to the electro-motor force plus internal and external
resistance. Ohm's law is that the strength of the current is equal
to the electro-motor force divided by the external and internal re-
sistance. *Stabile* application of a continuous current is where the

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poles are first applied in one place, then the current turned grad-
ually on. *Labile* is where the galvanic or interrupted currents are
set up first, then the poles placed to the parts to be treated and
constantly moved from one *nodal* [1] point to another.
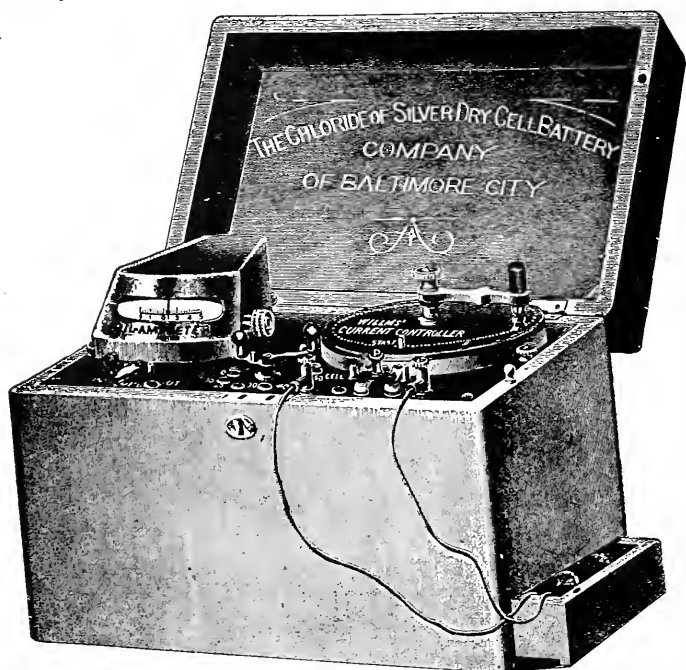
Fig. 21.—COMPLETE GALVANIC DRY-CELL BATTERY (with milliampèremeter and rheostat).

The current that produces the greatest contraction of the mus-
cles with the least pain is always applied.

A "dry" cell is one in which the conducting medium in the
battery is not a liquid, and, while convenient, is not to be depended
upon as is the "wet" cell.

The *sinusoidal current* is one that comes in therapeutics be-
tween the galvanic and faradic currents. It is a current of greater
intensity and less in quantity than the galvanic current, and is
the result of insertion of a series of magnets in the galvanic cur-
rent. It is used as a stimulating agent in cases of extreme atrophy
of muscles, etc.

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[1] Nodal points are points of constant potential in a muscle. Motor points are points in a muscle more irritable than others.

FARADIC ELECTRICITY

The faradic current is the so-called *induced* or interrupted current, and is generated from a cell in a like manner to the galvanic current as described. It is, however, so modified by the interposition of a coil of fine iron wires as to change the character of the electricity by " induction " to great electro-motor force; but it has little chemical power compared to that of the continuous or galvanic current, because the fact of breaking a current with the inductive coil changes the manner entirely in its action upon the human body. This current is simply used for the exercise or development of muscular tissue because it produces contraction of muscle. These contractions are either *rapid,* when the rapid inter-

Fig. 22.—Combination Dry-Cell Galvanic and Faradic Battery.

rupter is used, or *slow,* as when the large lever is placed within the current and by magnetization is caused to swing to and fro slowly.

In using this kind of electricity pain is not produced in the patient so easily as it can be produced by the galvanic current; at least, not of that burning or chemical character, as has been mentioned. In fact, we do not think of using the word *qualitative* in relation to faradic electricity, since *quantity* of contraction and but little or no qualitative symbolism maintains; so little, indeed,

that it is not necessary to tax the memory with the very small
amount of difference between contractions made when the positive
faradic pole or the negative is placed over the muscle stimulated;
since, too, the pole is changed through the fact that the direction of
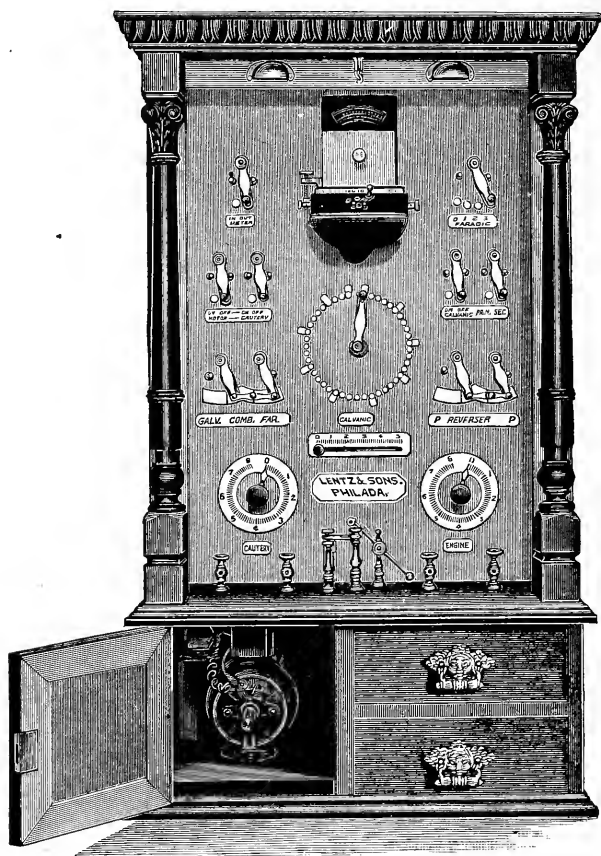
Fig. 23.—COMPLETE ELECTRIC CABINET (with dynamo alternator).

the current is changed with each interruption caused by the act
of induction itself.

USES OF FARADIC ELECTRICITY

The faradic current is used, as has been inferred, for the de-
velopment of the muscles. The " rest treatment," consisting of
massage, isolation and rest in bed, full feeding and electricity, is

aided by faradism very much in the efficiency of the fullest treat-
ment and benefit to the large majority of cases.   The application
of faradism to the human body, as typified in this course of treat-
ment, we shall now indicate.   This consists of about forty-five
minutes' application in the following fashion: The patient should
be in repose and treated at about midway between the meals, prefer-
ably in the morning or afternoon or early evening, all depending
upon the time of giving massage, the two separated widely in the
waking hours.   The room should be at about 75° F. in the ma-
jority of instances of neurasthenia, or varying with the idiosyn-
crasy of the case; the point being, of course, to prevent "catching
cold."   Having the patient placed on one side and thoroughly re-
laxed, the *rapid* current is applied to the feet by metallic electrodes
well covered with absorbent cotton.   This will not take more than
five minutes, and will produce an agreeable sensation of tingling
to the patient, warming the extremities at the same time.   Then
the *slow* interrupter is placed, and the two poles are applied over
the bellies of the extensor and flexor muscles of the leg alternately,
producing in each separate group well-marked contractions at the
breakings of the current.   This will take five minutes more.   The
thigh is treated in the same way, producing again as strong con-
tractions of the muscle as possible without causing pain, the guid-
ing principle being *never to produce pain* in administering any
form of electricity.   The other limb is treated in the same manner,
the patient being turned on the opposite side and the treated limb
being wrapped in a blanket for protection.   This will have con-
sumed about twenty-five minutes.   The upper extremities are
treated in like manner, and you will find the current much more
easily perceived here by the patient.   The agreeable sensation given
to the hand by the *rapid* current is most soothing, while the con-
tractions induced in the forearm muscles, then in the arm and
shoulder girdle musculature will have caused a sense of warmth
and stimulation to the whole of the members.   Now apply the cur-
rent for about twenty minutes upon the upper members—a shorter
time than applied to the lower limbs, but quite as efficient, however,
since the upper extremities and their muscles are much more easily
manipulated.   The blanket being folded across the chest protects
this part of the anatomy, and while a cover also remains across
the lower limbs (the patient lying supine), the broad flat muscles
of the abdominal walls are next treated.   Then the patient can be

turned over on the abdomen, the erector spinæ group of muscles are given a few moments' contraction or exercise, when the case will have been fully treated. The patient is now well covered, with the blanket kept next to the skin, and perfect quiet being insisted upon, he is apt to fall off into a delicious slumber.
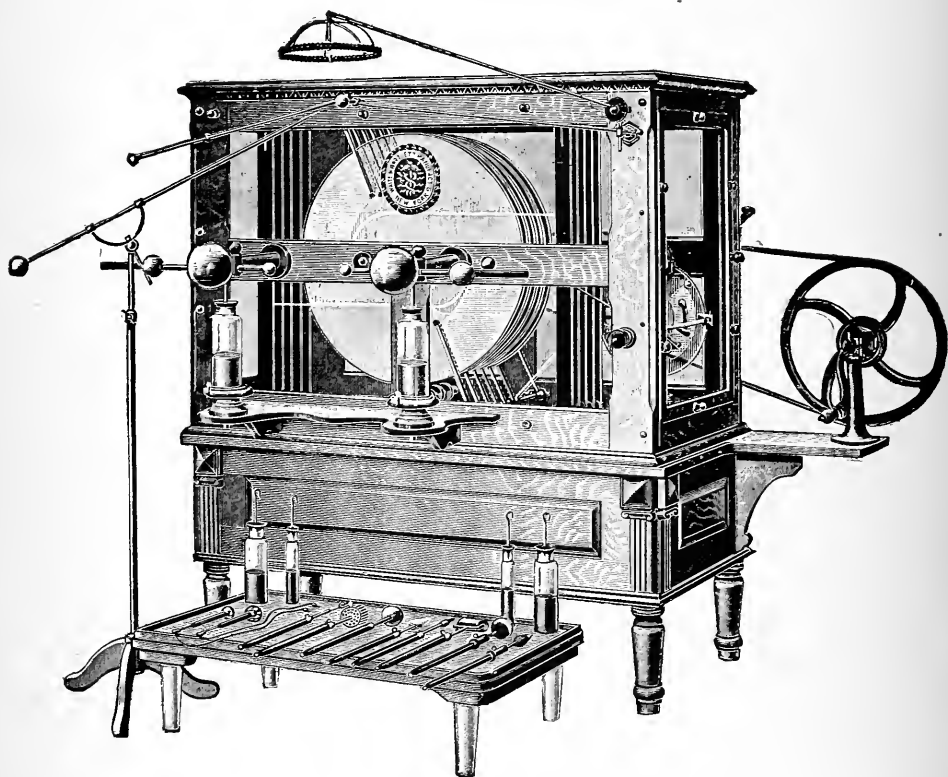
Fig. 24.—Static Machine and Complete Series of Electrodes.

Thus the patient gets objective exercise without calling upon his subjective state—i. e., the central nervous system—to furnish force from his own vitality, which is *needing* the rest. It must be remembered we are describing the typical case, and as with massage, exceptional instances occur which for some reason in an hysterical or excitable neurasthenic electricity will *not* be well borne. It should be stated also that the felicity and facility with which the faradic current is applied depend upon the amount of

practice and experience gained from persistent effort to do the thing well. With this ease of execution you will find the time of application can be cut down very much, and with the same efficacy to the patient.

The *faradic brush* attached to the negative pole to produce irritation is another tonic method of applying the induced current, useful in such symptoms as hysterical anæsthesia. The skin should be dry when this is employed.

FRANKLIN OR STATIC ELECTRICITY

This electricity is generated through friction, as of a revolving wheel upon leather or wool. It is a current of very low electromotor force, but with great intensity, and is used simply as a stimulant to arouse better circulation, such as in chronic rheumatism, old cases of hemiplegia, lumbago, and in chronic articular disease with joint pains. It is also a most active tonic, and a spark passed up and down the spinal column—the patient being placed on the insulating stool for three to five minutes—serves mightily to awaken nerve energy in one who is neurasthenic or in ephemeral nerve-tire from whatever cause.

Before giving this current especially should the patient be warned else he might interpret the snap of the spark as something serious, whereas it is so rapid and of such a minimum quantity that there is no danger whatever, and, indeed, burning of the clothing or even of a piece of paper placed between the poles will never occur. You regulate the amount of current to the pleasant sensation produced in the patient. This is done by sliding resistance rods more closely together, thereby lessening the resistance which the body itself would otherwise have to perform, and, of course, with greater discomfiture. The static breeze is a milder form and can be used upon the scalp for neurasthenic, helmet-like headache. (See Fig. 24.)

HYPNOTISM

By hypnotism (from Gr. *hypnos*—sleep) we refer to the induced sleep or state of sleep produced in a subject by a second person.[1] It is more easily induced in persons or individuals the subject of

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[1] Some authors claim that there is a self-induced hypnotism, but this will be found to be the result of suggestion immediate or remote from a second person in every instance.

highly nervous temperaments, or actually suffering from some func-
tional nervous disease. Some psychologists contend that hypno-
tism can be induced in the best minds as readily as in those persons
referred to, but this is not my experience, although I have seen
a very intelligent man hypnotized after a prolonged effort. In
medicine, hypnotism should be used entirely as a curative agent.
The school of Bernheim and Charcot have developed this form of
therapeutics more than any other; and while there are a number
of different stages given by certain authorities on this subject, it is
sufficient to here define only three stages of the hypnotic state:
(1) That of lethargy, in which the patient's mind is obtunded.
He breathes easily and the circulation is quiet. In a few moments
in the typical instance of hypnosis the patient will pass into (2)
catalepsy, which latter consists of the lethargic state plus muscle
rigidity, and from this the subject gradually passes into (3) som-
nolence, when he breathes deeply and is apparently sleeping in a
relaxed state. These three subdivisions are not accurately defined
in the majority of subjects, one predominating over the other,
depending upon the idiosyncrasy of the case. The *methods* of
inducing hypnotism are many, but all depend upon the same phe-
nomena—namely, absorbing the patient's interest entirely in one
direction, having prepared him by the suggestion that he should
think of nothing but sleep and at same time try to sleep. The
holding of revolving mirrors before the eyes or any bright object
for fixation will be the instrumentality used. The physician, of
course, must have the bearing of confidence to impress the patient,
and the subject should be told that nothing will be done save the
desire to cure the given ailment. It is well to have the person
in a quiet room and to fix the eyes in a strained position so that
they soon tire. Thus the conscious mind is held in abeyance and
the subconscious mind alone predominates, the subject passing
under the control of the manipulator. It is desirable to place the
patient in the third stage, if possible, before attempting sugges-
tion, although in difficult subjects suggestions for cure may be of
avail even when the patient does not pass beyond the lethargic
stage. The suggestion for cure should be made while the patient
is hypnotized, and for practical success should not be too dogmat-
ic, as, for example, in case of hysterical palsy. In this way the
doctor has better control and the patient is not disappointed;
moreover, the physician will more likely be able to succeed in the

hypnotism of the subject at another time if he fails in the first attempt. Indeed, in many patients or subjects that are really susceptible to hypnotism, it will be found that it must be resorted to at different times before suggestion for cure can be pressed upon them. The *post-hypnotic suggestion,* so called, which consists in the endeavour to induce in the subject hypnotized the doing of certain acts or the producing in them of certain sensations long after the hypnotic state—thus, a suggestion that tobacco will be unpleasant to the tobacco fiend—is a possibility.

Hypnotism has a very limited range and should be used as a last resort in cases where the ordinary mental influence of the physician does not avail. It is especially of value in local hysterical palsies, either sensory or motor, and is distinctly of less value as the functional paralysis is more general. Cases of hysterical hemi-anæsthesia I have never seen benefited by its use.

THE REST TREATMENT

This method of treatment, devised by S. Weir Mitchell, is of especial value in neurasthenia and hysteria.

Its *essential features* are isolation, absolute rest, diet, massage, electricity, and the personal influence of a good nurse.

Isolation from the patient's family and former surroundings is essential. The *diet,* if the digestion is bad, should be entirely of milk. Otherwise easily digested solid food may alternate with it.

Rest must be absolute in severe cases; even sitting up in bed should not be allowed. The duration of the treatment should be from four to eight weeks.

The following schedule, as given by Dr. J. K. Mitchell, will illustrate the method:

7 A. M.: Cocoa; cool sponge-bath, with rough rub and toilet for the day.

8 A. M.: Milk. Breakfast. Rest for one hour.

10 A. M.: Eight ounces peptonized milk.

11 A. M.: Massage. (See previous section.)

12 N.: Milk or soup. Reading aloud by the nurse.

1.30 P. M.: Dinner. Rest one hour.

3.30 P. M.: Eight ounces peptonized milk.

4 P. M.: Electricity (general faradization).

6 P. M.: Supper, with milk.

8 p. m.: Reading aloud by nurse for one-half hour.

9 p. m.: Light rubbing by nurse with drip-sheet.

Eight ounces malt extract with meals; tonic after meals. Eight ounces peptonized milk with biscuit at bedtime, and a glass of milk during the night, if desired.

This may be modified in various ways according to the symptoms. If a mild case, the patient may be allowed to sit up for a few hours, or even go out for a drive.

Object of the " Rest Treatment."—Of which rest is the essential factor, is (1) to secure repose and time for recuperation from exhausted nervous states, and this is assisted by (2) isolation from friends and other sources of cause for expenditure of nerve-energy; then comes (3) massage and electricity, both agents to mechanically exercise the muscles, to hurry the blood and lymph streams on, and to stimulate metabolism without using up the patient's little reserve force. To use Dr. S. Weir Mitchell's apt phrase, " massage and electricity deprive rest of its attendant evils." Imagine any one in health lying abed for twelve weeks or more without this excellent energizing agent. He would more likely waste than gain in nerve or muscle tone, and even if his weight should possibly increase during the time, it would be in flabby tissues, the majority of which would be only deceptive evanescent fat. Fat in itself is a burden, not a gain, to the individual. Better be " lean " and hardened, as the athlete or thoroughbred, than to have excess of the so-called *panniculus adiposus*.

ON THE CARE AND TREATMENT IN CONVALES-CENCE FROM FUNCTIONAL NERVOUS DISEASE

There is no department of neurology more neglected than the proper understanding, or at least if appreciated the neglect of sufficient advice to convalescents from serious disorder of the nervous system; and in this place we shall devote especial consideration to functional diseases, those so considered in the absence of accurate scientific data to throw the light of a definite pathology upon many cases of them. Therefore those diseases to be discussed shall be naturally enough neurasthenia, hysteria, and chorea—the author admitting, however, that there are many cases secondary to some physiologic perversion or to an irritation; still, as it is always difficult to determine the *fons et origo*

mali, the majority of cases are usually set down in the text-books as functional. As it is not intended to be specially scientific as to ætiology, and as the resultant disease (whether functional or due to primal causes other than the ordinary overwork, mental strain, etc.) is the same, the treatment of convalescents from either essential or from secondary neurasthenia would be in large measure alike. Dismissing at once this subject of ætiology—not because of its lack of importance, for we purpose insisting upon the keeping clear of all causes for the development of a secondary condition in the beginning or at any stage—we shall formulate certain principles to be obeyed in the management of cases where diagnosis of the diseases under discussion has been established; in other words, the care of the patient after the malady has fully developed and has been treated successfully towards the happy issue of cure, which latter can be maintained *only* by the principles that we shall attempt to lay down in this contribution. We will include therefore, in this practical treatise, suggestions for the betterment of the person who is called by the physician—for the want of a better name—"generally nervous," the result of previous functional nervous disease.

Mental Care.—In the first place, these patients must be schooled or educated in self-control. This may seem to savour a bit of charlatanism; but when the profession learn to better appreciate that there is really a psychologic basis, a suggestion, in this form of therapeusis, there will be less chance for Christian Science and other fads, with more adherence to true medicine, so that the proper balance may some day be reached, and the modest medium of common sense more generally prevail. Nor will there exist, at that happy time, any basis for the rather trite slur at the social fabric which "Mr. Dooley" has recently expressed, in effect, that Christian Science believes there is no disease, and physicians believe that everything is disease.

A certain amount of time will be required therefore, by the alienist and neurologist, in explaining away, to intelligent people, certain fads with which they have become infected; and while the task is a hard one, results of treatment will show a fair enough proportion of success, and the doctor will at least be given credit for making the endeavour to rid society of these baneful influences. Of course, if you are dealing with a crank, diplomacy will be shown by dropping the subject. As it

has well been expressed by some one, " You can take a crank by the hand, but cannot lead him on." With these few words, we shall dismiss the somewhat mysterious side of medicine, so essential to success, however, and pass to more scientific truths—the care of the human body physically, the better understood and more easily explained matter.

Physical Care.—Most patients, after an attack of acute or prolonged functional disease, feeling the burden lessened and cure set in, will, in their exhilaration, and from the fact that the nervous system does not feel the insult at first, go back to the usual method of life—of strain and overwork—with a zeal that will usually precipitate disaster if persisted in; so that, as regards rest, the convalescent should be taught responsibility in the care of himself. If he refuse to carry out our directions, the result cannot be blamed upon his physician. These are they who pass from one reputable man to another, without any permanent good results. This is only within the bounds of reason, and does not necessarily require scientific demonstration, valuable as that may be. It is an axiom of nature that rest should follow after the central neurones have once been depleted, physiologically or pathologically. Even in cases of hysteria with full muscular vigour, I doubt if overuse of the muscular system will but prevent fullest recovery. In the cases of hysterical palsies, it is necessary, however, to encourage exercise to a limited degree in order to give confidence to the patient, as well as for development of the wasted muscles. Thus, the most desirable forms of exercise during the late, ultimate convalescence from neural maladies are in a general way to be made different from the exercise obtained by the patient in his usual routine of life; and here again the psychologic element must enter prominently as the reason for this betterment. Exercises, therefore, that keep the patient away from his usual line of thought, or prevent him from thinking about self, are to be enjoined. And if the patient can be kept off his limbs, it will be more advantageous for rest of the central nervous system, and thus recreate that reserve force which has become wanting in them. So that rowing, canoeing, horseback-riding will be much more valuable than golfing, tennis-playing, or bowling.

Adjuncts and Medication.—It is important to treat symptomatically organs that are disturbed in function during convalescence from the diseases under discussion; thus, in cases of gastralgia or

of supersensitiveness of the gastrointestinal tract, it may be neces-
sary to treat the mucous membrane. In these cases, we have a
neurosis of the vagi nerves and of the abdominal sympathetic
system.

In the case of Mrs. W., a woman of delicate constitution, aged
thirty-six, under the writer's care, suffering from essential neuras-
thenia, the result of society indulgence, one of the most annoying
features in convalescence was subjective sensation of distress in the
abdomen. Three years previously, this had assumed a form of
distinct gastralgia. The use of intestinal antiseptics, as small
doses of salol, also the addition as a digestant of ox-gall, gr. i,
and extract of gentian, gr. $\frac{1}{8}$, a half-hour after meals, acted effi-
caciously, and the patient has gone on to complete cure after a
prolonged treatment which had not entirely restored her equilib-
rium when she left for the country. It was this little addition
of a digestant that capped the climax towards cure. She gained
fifteen pounds in four weeks.

Another case of Miss P., aged twenty-five, a very intelligent
young woman, was a most persistent case of neurasthenia, greatly
benefited by intestinal antiseptic treatment in addition to a semi-
rest cure. In this case large amounts of indican were found in
the urine.

Another adjuvant in the treatment was *high flushings* of the
colon with a solution of lime-water, and it was interesting to see
a facial acne disappear with the general improvement of the case,
and especially after resort to the measures mentioned. The reten-
tion of toxines in the system is undoubtedly a factor in preventing
many cases of neurasthenia from reaching fullest health; until
this detail is carried out, the patient will never become well. The
writer has seen good results in the treatment even of epilepsy by
means of antiseptics and flushing the intestine with copiously pure
water between meals.

Apropos of this subject, the reader is also referred to an article
by Morris de Fleury in the Bulletin Général de Thérapeutique,
November 23, 1900, in which he gives tables showing, in the Ex-
cretion of Urine in Neurasthenia, the amounts of uric acid, urea,
and phosphates; the earthy phosphates being greatly in excess
of sodium and potassium. Indican or skatol was found 73
times out of 100. The coefficient of oxidation was decreased
55 times out of 100; the increased acidity of the urine and density

of the same, 59 times out of 100 cases recorded. Such data as this goes to prove the wide-spread disturbance of metabolism in functional disease. An investigation [1] by the writer has been for determining the same conditions.

The treatment, therefore, will more and more depend upon the exact knowledge obtained through the physiologic and pathologic study of chemistry, it seems to us, for the permanent cure and prevention of these ubiquitous diseases; so that while the *gastro-intestinal tract*, the great laboratory of the body, may be secondarily affected in neurasthenia, it is essential to treat the *symptom* as well as the *fundamental* condition of nerve-cell exhaustion. Frequently peptonization of the large quantities of milk taken will be of great value in aiding hypernutrition. The skilled use of massage is also a measure which will help metabolism in the muscle itself, as well as by mechanically forcing on waste products and giving vigour to the circulation; for frequently, in these cases, the cardio-vascular system is not doing the physiologic duty required.

Excitement of an unusual nature should be strictly prohibited in cases of neurasthenia, and this is especially more important in the convalescent from chorea. In the latter disease, even pleasurable excitement may be sufficient to cause irritation, and relapse to follow. A very marked instance of this condition of things was shown in the case of a minister's daughter, aged fourteen, coming under my care on November 6, 1900. She had been continuously in choreic movements since January, 1900, having been somewhat improved in health by September, 1900, when she started to school and had a relapse. The slightest pleasures she enjoyed were sufficient to make her worse. The child was anæmic, complained of vague pains in the lower extremities, and was greatly emaciated. The use of Blaud's pill, 5 grs., three times a day, of salt sponge-baths at night and of arsenic in small doses, and the insistence upon the use of woollen underclothing, by July 3, 1901, eight months after beginning treatment, brought her to a timely cure —to health she had not known for many years. This case is detailed to show that chorea is no slight disease to deal with, and that it should be studied in all of its aspects as to hygiene for the patient, if health is to be restored. It may have been that previous

[1] Uric Acid Excretion in Neurasthenia, Transactions of the College of Physicians of Philadelphia, vol. xxii, 1900.

failures in this case were ·due to a lack of hygienic precautions being insisted upon to the parents, who were perfectly willing and did carry them out when so forcibly directed, of which they are the greatest appreciators to-day.

CLIMATOLOGY OF NERVOUS DISEASE

Under *climatology* come temperature, humidity, winds, purity of air, rarefaction of air, sunlight, electricity, soil, woodlands, and social surroundings. The therapeutic climate is one which improves functional activity or increases the resisting power of the organism. Climatology, the medical geography of climate, is to climatotherapy what materia medica is to therapeutics. The main use of climatotherapy is as an adjuvant to ordinary therapeutics. Dr. A. Manquat, of Nice, has given the best authoritative study of this important subject.[1]

Difficult though it be, certain fundamental facts must surely exist and are being gradually worked out. The writer has made some studies [2] upon the climatology of neurasthenia, finding that one of high winds or in an altitude above 2,000 feet, or in damp, low countries, is bad for this disease. Moderately high altitude, 2,000 feet, is, on the contrary, desirable in sclerotic cord diseases, since decreased atmospheric pressure favours better circulation.

More elemental facts to be recalled are that air at sea and at high levels is purest. The temperature above sea-level diminishes about 1° F. for every 300 to 400 feet, and is less the drier the air. Temperature of air varies less near sea-level, and is less in the southern hemisphere. The higher the elevation and the colder the air the less moisture it contains. Ozone and electric influences of mountain countries are apt to irritate nervous people. Weber speaks of *marine, low-level inland* and *high-level inland* as the great divisions of climate. Warm marine climates and sea voyages on calm water are best, perhaps, of all for neurasthenics. High climates are best for anæmic cases. The Riviera, low Colorado, Canada (in summer) are the most desirable climates for the nervous invalid. Inland Maine is also of value.

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[1] Bulletin medicale, November 27 and December 14, 18, 1901, and January 1 and 8, 1902.

[2] Climatology of Neurasthenia, Medical News, January, 1901, and Nervous Cardiac Symptoms due to High Altitudes, February 14, 1903.

CHAPTER V

SYMPTOMATIC DISORDERS

VERTIGO

By vertigo we understand a disturbance of consciousness, characterized by a feeling of objects moving (objective vertigo) or of a sensation of the person moving, which is termed subjective vertigo.

Causes.—The causes of vertigo may be classed somewhat like those of headache: (1) Hæmic—anæmia, hyperæmia, etc.; (2) toxic—tobacco, alcohol, lead, etc.; (3) arterial sclerosis; (4) acoustic nerve irritation; (5) neuroses—neurasthenia, epilepsy, etc.; (6) reflex—ocular, gastric, organic, as organic brain disease; (7) mechanical causes, as swinging, sea-sickness, car sickness.

Symptoms.—They usually come on suddenly, and last but for a few moments. The patient may have the sensation of rising or sinking, or objects moving or whirling about (the objective form), or the patient himself may feel as though he was revolving or to rise and fall (subjective form). There is a sense of alarm and a feeling of faintness. Cerebration is disturbed and ideas are confused. There may be nausea or vomiting. The patient usually totters, and sometimes falls. It may be due to certain lesions of the cerebellum and its peduncles, or of the labyrinth of the internal ear. It is then usually associated with forced movements. The characteristic of vertigo is that it is relieved when the patient lies supine. This is the opposite condition in a case of headache. (See section on Headache.)

A large proportion of the vertigoes met with are due to disease or irritation of the eighth nerve and its centres. The more common cause is a lesion of the labyrinth of the internal ear. Vertigo, therefore, is a symptomatic disease. If the blood is debilitated, anæmia, or if the patient is plethoric, hyperæmia, the symptoms of this affection will come hand in hand with the vertigo.

If the disease be due to arterial sclerosis, which produces a localized anæmia of the brain, the vertigo will then be associated with symptoms of cerebral arterial sclerosis. In some of these cases the kidneys are also affected in the process, and we have the symptomatology of the red-granular kidney, low specific gravity to the urine, decrease in the quantity of urea, etc. If associated with neuroses, we have the characteristic symptoms of these several affections, such as neurasthenia and hysteria, and the vertigo will be apt to present exacerbations commensurate with the paroxysms of the several diseases, or else the vertigo will be more exaggerated sequent to aggression of symptoms of the neuroses mentioned. The reflex vertigoes will be associated with the organ that is particularly involved, as the cause of the irritation. Thus a patient suffering with astigmatism would complain of the eyes, as a rule, but not always so, however. The patient with gastric vertigo would have gastric attacks or crises. This is more usual in persons about middle life. From organic brain disease the reflex vertigo would be associated with some pathologic process going on within the cranium. The mechanical causes giving rise to vertigo will be easily enough determined by the careful observation of the physician. Toxic vertigoes are associated with sepsis in different parts of the body, or are from drug poisons, such as tea, lead, coffee, etc. Auto-intoxications will be associated frequently with the finding of indican in the urine; the gouty with secretion of urea, uric acid, and alloxuric bodies, and other end products.

Diagnosis.—The differential diagnosis of vertigo would depend, as above indicated, by noting the symptoms of the several diseases associated with, preceding, or following the attack.

Prognosis.—This depends entirely upon its duration and the ability to cure the cause. Hæmic and toxic vertigoes will usually recover if treated early. Those cases due to arterial sclerosis are very bad as to the prognosis. Those due to neuroses are also difficult cases to manage. The mechanical vertigoes will subside with abatement of the cause.

Treatment.—Quietude is an essential. This will depend entirely upon the cause. If there be anæmia, hæmatics should be administered, such as tincture of chloride of iron and Bland's pill. If due to plethora, venesections, or better, wet cupping or the application of the natural leech, will often prove of great value. The use of potassium iodide as an alterative and sorbefa-

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cient, continued in small doses, often produce marked ameliora-
tion of the symptoms on any case. Nitroglycerin $\frac{1}{100}$ gr. t. i. d.,
or spt. of glonoin, 5-minim doses. If ear disease is present, the
case should be referred to a competent aurist for the proper diag-
nosis and treatment of the condition.

The patient should lie supine during an attack.

INSOMNIA

This is sometimes called sleeplessness, and is given to those
conditions of insufficient or restless sleep or to the entire absence
of sleep for a long time. Certain persons present idiosyncrasies
as to the amount of sleep required. Napoleon required but a few
hours out of twenty-four. It is said that some people can go with-
out sleep for indefinite periods, as long as two or three weeks at a
time, cases being on record of much longer times of the waking
state, inflicted as a punishment. Eight hours in twenty-four is
the average for an adult; children require more.

Causes.—The male sex suffers more than the female sex, no
doubt due to employment being more of a mental nature in men
than in the majority of women. Labouring classes are less liable
to insomnia than those engaged in business or professional pur-
suits. It is frequently hereditary, no other exciting cause existing
that is known. It is also seen in certain diseases, such as gout and
lithæmia, etc. Quite probably it is the result of auto-intoxication,
the absorption of certain ptomaines or leucomaines, or other dia-
thetic by-products. Diseases of the cardio-vascular system may
lead to insomnia. Poisons may also cause insomnia, such as are
seen as the result of syphilis, malaria, chronic nephritis, or other
infectious diseases, lead-poisoning, etc. Those cases which are
not due to definite lesions are designated functional; for instance,
the insomnia of neurasthenia and vaso-motor disorders.

The *time* of the sleeplessness varies. Some persons cannot
sleep during the early hours of the night, and others will awaken
at a certain time and cannot sleep thereafter. In children it is
accompanied by much mental and physical disturbance, such as
dreams, physical or mental excitement.

The patient is also seen to be irritable from the loss of sleep,
and this is much more exaggerated in children than in adults.
Insomnia from neurasthenia usually presents a tumult of thoughts

passing through the mind which seem to prevent sound sleep; or in them sleep is very superficial, the patient being aroused very easily and frequently imagining that he was partially awake during the sleeping period. In this form the patient wakes easily and afterward feels "draggy," as though he had had no rest at all, and frequently describing the sensation as though he was weaker after his limited sleep than before the time when he went to rest. In the insomnia of adult insane, there is a still greater degree of restlessness. A symptom described by Weir Mitchell frequently occurring just at the *prædormium* is a sudden jerking of the body, which arouses he patient just when he was passing into a doze.

Pathology.—This seems to be as yet unknown. It is very likely, however, that there is a variation in the calibre of the cerebral vessels, causing congestion, due to a weakness of the sympathetic system. "Retraction" of the cerebral neurones is also given as the "neurone theory of sleep." The pathology of symptomatic insomnia, due to organic disease, would be the pathology of the disease in question, as a chronic meningitis, cerebritis, gummata, etc.

Treatment of essential insomnia, so called, lies often in the treatment of some general disorder, such as hysteria or neurasthenia. If anæmia, lithæmia, uræmia, or other toxic influences exist, they must be removed by the proper remedies for these conditions. Measures for the relief of the symptoms themselves should be adopted, and the patient should get rid of worry as much as possible. The use of the constant cold douche to the spine from three to five minutes, followed by cold sponging of the body, usually produces sufficient reaction to cause a dilatation of the superficial blood-vessels to induce sleep. The overuse of the hot bath is to be decried, since it is apt to weaken the patient, and by not producing proper reaction to the surface will allow stagnation of the blood in the meninges. The methods of counting or other monotonous occupations will sometimes avail, but these are of doubtful efficacy, and should be substituted by other forms of occupation. The patient should live in the open air as much as possible, and particularly in those localities where dry winds prevail and at low altitudes. Drugs used with caution are bromides, 10 to 15 grains at bedtime or in combination with chloral hydrate, 5 to 10 grains, being cautious to guard the heart with

the use of the latter drug. Trional or sulphonal in 15-grain doses
in hot milk are the best hypnotics. Hyoscyamus in some instances
is efficacious. Paraldehyde in drachm doses is a valuable hypnotic
in insomnia due to alcoholism. Camphor, opium, and the evanes-
cent drugs, as asafœtida, musk, valerian, etc., are all of value in
hysterical or neurasthenic insomnia. The use of massage and
Swedish movements at bedtime are measures that are of great
value, and particularly the effleurage to the face, thus producing a
quiet effect through reflex action upon the cerebral cortex.

NEURALGIA

This is a type of pain, shooting in character, occurring along
the course of a sensory nerve, and functional or toxic in nature.
There are different varieties of this disease, as follows: trigeminal,
brachial, sciatic, cervical, intercostal, anterior crural, and visceral,
etc. *Reminiscent* neuralgia is that which exists after the cause
has disappeared. It is at times a monohysterical sign.

Causes.—Common predisposing causes of this condition are
early adult life, debility, and hereditary predisposition. The *char-
acter of the pain* is sharp and shooting in nature, and frequently
accompanied by tender spots or points over the exit of the nerve
from a canal (points of Valleix), although firm pressure over the
site of the pain will generally give relief. Vaso-motor and secre-
tory symptoms may occur.

Facial Neuralgia.—This is induced by cold exhausted condi-
tions, and is more frequent in the female sex. It has a distinct
hereditary tendency. The attacks come on at irregular intervals,
and involve some one or all the three branches of the fifth nerve.
The pain is sharp-shooting and paroxysmal.

Treatment.—This consists principally in the protection of the
surface and the building up of the system, and for the attack the
use of analgesics, such as phenacetine and antipyrine. The appli-
cation of warmth to this in the shape of hot water is sometimes
a means of relief. The use of cod-liver oil, to build up the system,
is a valuable adjunct. Aconitia in $\frac{1}{100}$-grain doses is of value.

Tic Douloureux.—This is a most severe form of facial neu-
ralgia.

Symptoms.—A sense of numbness, occurring at the site of the
branch particularly affected, such as the superior maxillary branch,
finally followed by dull pain, which soon amounts to acute suffering

and rather spasmodic in nature. In the height of the paroxysm the patient is in most exquisite suffering, all the branches of the fifth nerve being involved. There may be an ephemeral blindness, occurring on the side affected. An attack may last from one to two hours to twelve hours or even longer. There may also be spasm of the facial muscles. The teeth may be very sensitive during or after an attack, and sometimes the pain is so localized in the teeth or maxillæ that the physician or patient may think the tooth is the real seat of the disorder, and for this reason many teeth are extracted. The frequency of paroxysms varies from once a day to once in several weeks, although some cases may go much longer. The exciting causes are worry, overwork, exposure to draughts, and dampness.

Diagnosis.—This lies between this disease and migraine, but the character of the pain in the two diseases is essentially different, and there is not the nausea and vomiting in the trifacial neuralgia as in sick headache. The type of pain which is induced by these paroxysms and may become chronic or hallucinatory is an example of reminiscent pain, and is a psychic phenomenon purely.

Prognosis.—As a rule it is unfavourable. The patient seldom gets entirely well after the disease has once been established. Some few cases, however, have been cured when treated in their incipiency.

Pathology.—This consists in subacute neuritis in the branches of the fifth nerve, also degeneration of the Gasserian ganglion in the worst cases.

Treatment.—In the treatment, examination of the teeth should be a point in order to determine reflex points of irritation. Any other irritants about the face should be eradicated. For the paroxysms of pain, the use of analgesics is indicated, as phenacetine combined with caffeine. Galvanism by placing the positive pole over the painful area is of service. At times a hypodermic injection of morphine will have to be given in order to relieve, although this should be in a guarded measure in the treatment. Cannabis indica is of some value in these cases when given in prolonged small dosage. Cod-liver oil and tonics, with the use of iron in anæmic cases, is very good treatment. Excision of the Gasserian ganglion is to be had as a *dernier ressort.*

Intercostal Neuralgia.—This is a form in which the pain is limited to the distribution of the intercostal nerves. This is some-

times designated pleurodynia. The pain is worse on movement of the side or in the ordinary respiration of the patient, so that it is frequently called " a stitch in the side."

Prognosis.—This is favourable.

Treatment.—This is the same as for other forms of neuralgia, plus the strapping of the side of the chest; or the use of counter-irritation by means of the use of the actual cautery will sometimes give relief. The so-called *herpes zoster* is the type in which we have a neuritis of the intercostal nerves and frequently an eruption, vesicular in character, along the course of the nerve. This disease seems to be in some cases infectious in nature, and is more frequently seen in childhood than in adult life, it also very frequently being bilateral.

The treatment of herpes zoster does not differ from that of other forms of neuralgia except that protection to the eruption of the part should be insured. This can best be done by means of oiled silk or cotton batting.

Sciatic Neuralgia.—This is neuralgia of the sciatic nerve. Some doubt the existence of pain in the sciatic nerve area without there being an active inflammatory lesion present. There are a certain number of cases in which the increased reactions and exquisite neuralgic pain, without tenderness on pressure, would lead one to suppose they were not inflammatory.

Prognosis.—This is, as a rule, good.

Treatment consists of splinting the lower extremity during the exacerbation of the disease. The use of the ordinary remedies mentioned above is indicated. The majority of persistent cases that do not respond to the treatment outlined are really of an inflammatory nature—a neuritis. (See section on Neuritis.)

Anterior Crural Neuralgia.—This is a neuralgia existing along the course of the anterior crural nerve, as its name would indicate. The pain shoots down through the outer upper aspect of the thigh towards the inner side of the knee. This sometimes is induced by lesions, as indeed is sciatic neuralgia in the pelvis, such as tumour, growths of bone. It is also designated *paræsthetic neuralgia* when the distress is of a burning sensation.

Prognosis.—This is good if the original cause can be relieved or cured, as through removal of a tumour.

Treatment.—This is the same as for the other forms of neuralgia, except that nerve stretching is singularly often curative.

Visceral Neuralgia.—Gastralgia is one of the most important forms. This consists of pain localized to the epigastric region. It bears no relation to the ingestion of foods particularly, excepting that it is at times relieved by taking of food. The disease is due to the irritation of the gastric filaments of the pneumo-gastric nerve.

Prognosis.—This depends upon the condition of the system. If metabolism improves the patient usually recovers.

Treatment consists in the application of heat to the epigastrium, the use of Fowler's solution in gradually ascending doses, given after meals (gtt. iij to x t. d.) and treatment of existent neurasthenia. The use of repeated blistering I have seen cure.

Neuralgia of Heart.—This consists in painful paroxysms about the præcordia, and accompanied by a subjective feeling of pending death. There is a sense of constriction, as though the heart was grasped in a vice. The lips become pale, the pain is most intense in the præcordia, of a constricting character, and frequently shoots down the left upper extremity. The patient becomes cold and clammy, and lies, through both fear and inability to move on account of the pain, in a position of fixity of the entire musculature until the passing of the spell, which may last from three to four minutes to an hour. The patient after the attack is greatly weakened, and it is some hours before the sense of fear and the pain subsides. Angina pectoris recurs without definite cause, although exercise may induce an attack.

Pathology.—Sclerosis at the orifices of the coronary arteries with narrowing and fatty change has been found.

Prognosis.—This is absolutely unfavourable.

Treatment consists in the use of amyl nitrate inhalations during an attack, nitroglycerin over continuous periods, and the relief of arterial sclerosis, if present, by the use of potassium iodide. Such patients should live a quiet life, never indulging in overeating or in stimulation. Mental strain should be avoided, since this is one of the patent causes. In the most serious attacks hypodermic injections of morphine may be necessary to relieve the pain.

HEADACHE

This is a condition of pain in the head. It is due to irritation of the fifth nerve branches. The characteristic of headache is that

it is usually made worse when the patient lies down—i. e., when the head is lowered, with the exception of anæmic headaches.

Causes.—Overwork, malaria, and other infectious diseases, poisonings from without, such as tea and various drugs; poisons from within, due to disturbance of metabolism, such as in gout and rheumatism, anæmia, and in diseases of the meninges. Finally, headaches are due to organic diseases of the brain, its membranes, or overlying structures. Some types of headache are:

Boring, often described as though a nail were driven into the vertex, the so-called lavus of hysteria.

Shooting or neuralgic headache, which is paroxysmal and extremely painful.

Constriction or helmet-like headache, characteristic of neurasthenia, consists in the sensation of constriction about the scalp with vague pain through the top of the head.

Finally, we have another type of headache, which is confined to one side of the head, and is called migraine or megrim or hemicrania.

J. C. Wilson gives as causes of headache:

1. **Reflex Irritation**—viz., ocular (eye-strain), nasal, pharyngeal, auditory, decayed teeth, reproductive organs (especially female), thoracic and abdominal viscera.

2. **Toxæmic.**—A. *Infections,* as acute infectious diseases; malaria.

B. *Incomplete* or perverted *physiologic-chemical processes,* or the defective elimination of waste, as uræmia, diabetes, gout, lithæmia, rheumatism, gastro-hepatic derangements, constipation.

C. *Action of drugs and poisons.* (*a*) Acute: nitrites, quinine, opium, alcohol, carbon dioxide, etc. (*b*) Chronic: lead, tobacco, alcohol, opium, tea, coffee.

3. **Circulatory Disturbances.**—A. *Passive congestion,* as by posture; tight clothing about the neck; pressure on veins by tumours.

B. *Active hyperæmia,* from excessive physical or mental strain; early stage of acute meningitis.

C. *Anæmia,* following loss of blood or the idiopathic anæmias, especially chlorosis.

MIGRAINE (Sick headache, megrim)

Migraine is a type of headache which is so distinctive in its phenomena as to warrant special discussion. There is a hereditary

predisposition very strongly manifest in this disease. First, direct hereditary predisposition to the disease itself is transmitted, second, where epilepsy or some other form of neurosis exists in the ancestry. The characteristic features are a periodic discharge of sensory impulse from the sensory nerves, producing pain. The attack itself is ushered in by hallucinations of sight, frequently accompanied by fortification lines, which are subjective phenomena of divergent lines appearing before the eye of the patient. Hemianopsia may develop. The pain is usually first manifest in the first division of the fifth nerve, and the pain is limited to one side of the head as a rule; hence the name of hemicrania. Soon after the onset of pain there is nausea and vomiting, the patient becoming very sick at the stomach and the pain reaching extreme intensity in from one to three hours, sometimes associated with a spasm of the muscles on the affected side of the head. The attack itself lasts from six to eight hours, when the pain gradually subsides, leaving the patient in a weakened condition, from which it takes him some days to recover full vigour. There is never loss of consciousness during the attack, but at times the exhaustion so closely simulates an abeyance of the mental state that epilepsy is with difficulty differentiated from migraine, some authors claiming that there is a direct relation between the two diseases. The attack of migraine comes on periodically, and the patient may have recurrences once a week or once a month, and in the female at the menstrual epoch, or less frequently. The attacks seem to be induced by overeating, auto-intoxication, and infectious diseases, such as malaria, etc.

Prognosis.—This is very indefinite. A guardedly good prognosis can be made in cases where there is not a history of direct heredity and in those of good constitution.

Pathology.—This has not as yet been definitely determined. So far as we know, it is a functional disorder, consisting of periodic discharge from the sensory cortical neurones, as already mentioned.

Treatment.—This consists of measures between the attacks and during the attacks. The emunctories should be kept in good condition, the liver and kidneys active. Any diathetic diseases should be treated, such as rheumatism, gout, etc. Carlsbad salts or Rochelle salts are of value to keep the bowels in a soluble condition and for the elimination of toxines. Salol, in 2-grain doses t. i. d.,

is of value here. For the attack itself, phenacetine is a valuable remedy, given in 5-grain doses, repeated three to four times each day, and if not effective can be substituted by other analgesic drugs, such as antipyrine. The use of tincture of cannabis indica is of value in some cases, and should be given in small doses, 3 drops t. i. d., gradually increased to the physiological limit. In the worst attacks hypodermic injection of morphine may have to be resorted to, but this must be guarded, else the drug habit may be brought about.

MÉNIÈRE'S DISEASE

This is a persistent neurosis in which vertigo is one of the most important symptoms. There is also irritation or disease of the eighth nerve or its centres. The inner ear is usually affected in the labyrinth. Deafness is progressive from the first.

Symptoms.—These are of the most exaggerated type of vertigo where other causes are excluded, excepting disease resident in the internal ear and where vertigo is associated with extreme nausea and attacks of syncope. In this form of vertigo, too, the progressive deafness and tinnitus aurium accompanied by forced movements, staggering gait, or absolute inability to walk, due to the dizziness in the worst cases, each time the patient arises from the horizontal position, are all diagnostic. The course of the disease is downward from the very first, the patient usually failing in general health on account of the extreme distress and inability to retain food. Deafness becomes profound as the disease progresses, and when complete deafness is present the vertigo ceases on account of the destruction of the auditory centres of equilibrium which have been irritated.

Diagnosis.—This is not difficult if the above points in symptomatology are carefully remembered.

Prognosis is guarded even in cases where the deafness is only slight and the disease not too far advanced. Usually the deafness becomes absolute when vertigo ceases and the patient is much better in general health.

Treatment.—The treatment indicated above for vertigo is employed. A drug which is harmful in the ordinary cases of vertigo, but is of value in these cases, is quinine. This is given in small doses and then increased until cinchonism results, the drug then being withheld for a time and renewed at intervals in fairly large

physiological doses. Opium has been recommended in this disease, but this should be used guardedly on account of the danger of the drug habit. Hydrobromic acid in the form of a syrup, given 30 drops a day, or the usual bromide salts in moderate doses, or salicylate of soda, 5-grain doses, t. i. d., will be of great service in these cases. Sinkler recommends ergot. Hirt recommends hypodermics of 10 drops of a 2-per-cent solution of pilocarpine. Bromides given in *large* doses (gr. xl, t. i. d.) have been recommended.

CHAPTER VI

CRANIAL NERVES

DISEASES OF THE CRANIAL NERVES

Olfactory.—This nerve may be affected either by *irritative* or *destructive* lesions. If the former, such as a tumour pressing on a nerve, it may produce besides the symptoms of the growth *hyperosmia*. Destructive lesions, as continued pressure of the tumour or a neuritis of the nerve filaments, would produce *anosmia*. Perversion of the sense of smell, or *parosmia*, may be a symptom in certain progressive diseases of the olfactory nerves or centres. A fracture at the base of the skull may be the cause of anosmia, as in a case of a woman seen under my care, where there were no other symptoms, and in which an injury seems to have been the exciting cause. A case reported by S. Weir Mitchell in his Clinical Lessons on Nervous Diseases, I have also had the pleasure of studying, but in addition to this the woman had hysterical stigmata, including amblyopia and disturbance of the sense of taste.

Diagnosis and Prognosis.—Disease of the olfactory nerve cannot be confused with other diseases, since the specificity of the condition is clear. The prognosis would depend largely upon the lesion discovered and the possibility of regeneration of the nerve in these cases.

Treatment consists in treating the cause.

Optic.—Affections of this nerve consist of functional amblyopias and organic diseases, such as choked disk, optic neuritis, etc.

Optic neuritis occurs in 85 per cent of the brain tumours, so that diseases of this nerve can be expected in that proportion in cases of brain tumour, and when found would be suspicious of the presence of a neoplasm within the cranial cavity. Atrophy of the optic nerve is either primary or secondary. The sclerotic diseases frequently have as a symptom atrophy of the optic nerve,

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such as in tabes; and secondary atrophy of the nerve would be that sequent upon neuritis, usually preceded by choked disk. *Hemianopsia* is a neurologic symptom, and is more frequently due to lesions in the tracts, usually unilateral. When the lesion is well back of the chiasm (as far as the primary optic centres), the centre for contraction of the pupil when light is thrown upon the retina is wanting, hence *Wernicke's* pupillary inaction will be found. If the lesion causing hemianopsia is back of the thalamus, the centre not therefore destroyed, the *Wernicke's* sign will not be present. The test for this important sign is made as follows: Throw a ray of light in a darkened room upon the unsound side of the retina; it will not cause the normal contraction of the pupil for the reason given; whereas, as soon as the ray of light impinges on the sound side of the retina, contraction of the pupil takes place immediately. This is the most delicate test, and has been by some writers thought impossible to make accurately, although admitting the scientific relation of the symptom to the disorder in question.

Other forms of hemianopsia are *binasal,* which condition is extremely rare, and is due to a double lesion, involving the outer fibres of the tracts as they pass to become a component of the optic nerves proper on their respective sides. *Bitemporal* hemianopsia is usually the result of a tumour of the pituitary body, which destroys the anterior fibres of the commissure, passing to the inner side of the retina. Hence in acromegaly where the pituitary is frequently found diseased that the symptom of bitemporal hemianopsia may exist. Hemianopsia may also be *horizontal,* as where a tumour or other lesion affects one-half of the nerve or tract above or below, destroying it or its function, and producing blindness in the upper or lower fields of vision. This, however, is a very rare affection (for illustration, see Fig. 19).

Hemiopia should be explained here, since it refers to the side of the retina blinded. For instance, right lateral hemiopia implies disease of the retina on the *right* side, whereas it means in terms of hemianopsia blindness in the *left* field.

Treatment.—Treatment of the diseases of the optic nerve consists in treating the underlying conditions when bearing upon nervous diseases. Optic atrophy seems sometimes to be benefitted by the use of electricity, passing 6 or 8 milliampères of the galvanic current through the head, with the negative pole at the occiput and positive placed over the eyelids. This done two or three

times a week (a three to five minutes application) will often be of some benefit to the patient. Conditions of errors of refraction, muscular unbalance, etc., are entirely in the province of the ophthalmologist, who should be consulted in all such cases. A disease in one of the optic nerves and closely allied to diseases of the nervous system is cataract, since this insidious blinding of a person may lead to general nervousness and even melancholia, which should be looked after in all such cases with the idea for relief through extraction of the cataract.

Oculomotor.—Paresis of the third nerve produces nystagmus, protrusion of the eyeball, and ptosis. There are two types of paralysis of the third nerve—one in which the *external* muscles alone are involved in the palsy and to which the name *external ophthalmoplegia* is applied, and second, where the *internal* muscles of the eyeball are paralyzed, designated *internal ophthalmoplegia.*

FIG. 25.—CONGENITAL NYSTAGMUS (mother also affected).

The causes are basilar meningitis, syphilis, and neuritis of the nerve and trauma as in some cases of fracture of the base of the skull. If the cause of the lesion is recent tertiary syphilis, the prognosis can be considered fairly good. If it is an evidence of parasyphilitic intoxication, the treatment is not of much avail. Instances of basilar meningitis are guarded as to the prognosis, since no one can tell how much destruction will result in incipient paresis of the nerve from the pressure that is produced.

Diagnosis.—This could not be confounded with any other condition, if the movements of the eyeball in relation to the anatomical and physiological condition of the eye are carefully studied, external strabismus being the most prominent symptom besides the ophthalmoplegia already mentioned above.

Treatment.—This consists in treating the original ætiological factor. Potassium iodide in doses of 10 to 30 or 100 grains

t. i. d. is usually one of the most valuable treatments if persisted in early in all acute cases. Later, after the absorption of the exudate has taken place, the use of strychnine, if generally indicated. If fracture of the skull occurred and the nerve was severed, there cannot be any hope for amelioration, since operation could not reach the site of the trouble without being the cause of death in itself.

Patheticus.—The palsy of this nerve is due to either neuritis, meningitis, or trauma. It causes paralysis of the superior oblique muscle; the eye is permitted to turn upward and inward. If the condition is dependent upon a neurological lesion, as indicated above, treatment would come under that of the neurologist, and would consist in the administration of iodides, and later strychnine. The ophthalmologist, as in all diseases of special nerves of the eye, should be consulted in order to determine any special treatment of the eye affection itself. Isolated palsy of the fourth nerve is rare.

Trifacial.—This is one of the most important nerves in the affections of same, from a neurological point of view; since diseases of this particular nerve produce more suffering and occur more frequently in neurotic individuals than in any other nerve. The cortical centre of this nerve is in the anterior part of Broca's convolution, the lower centre being in the floor of the fourth ventricle. The lesions producing diseases of the fifth nerve are either central or peripheral; excepting in some few cases of cortical diseases where the lesion is supranuclear. There would not be the reaction of degeneration in any degree of destruction of this centre, whereas, if peripheral parts of this nerve are involved, degeneration of the branches would occur. This is a distinctive point in regard to all the cranial nerves, and must be carefully studied in making diagnosis of a given case. Neuralgia of the fifth nerve is the most common disease affecting it. Tic douloureux has been mentioned under the head of separated diseases (see Neuralgia, p. 117). The simple neuralgia of the fifth nerve would be looked for in the different branches, acccording to the site of the disease, which has already been mentioned under the proper heading (see p. 117). It should be especially noticed in diseases of the inferior maxillary branch, it having a motor fibre as well as motor symptoms.

Abducens.—Paralysis of the sixth nerve produces internal strabismus. This is frequently due to neuritis, not an unusual lesion.

The treatment would be for the neuritis, and further, the case should be referred to the ophthalmologist for an operation upon the muscles affected if degeneration has occurred.

Facial.—This is affected from supranuclear or infranuclear disease. In disease of the cortical centre, which lies in the lower Rolandic region, there will be primary facial, spasm and secondary paralysis without degeneration. Here the lesion is generally unilateral, with the spasm or paralysis of the muscles opposite; the corrugator super-cilii muscle not being affected. Nuclear lesions are generally bilateral, usually hæmorrhage or gummata involving the floor of the fourth ventricle. The symptoms would be primarily spasm of the muscles, and, finally, paralysis with wasting and *reaction of degeneration*. The peripheral lesions of the seventh nerve, which produce symptoms of seventh-nerve disease, are, first, the *intracranial*; second, *intra-osseous*; and

FIG. 26.—FACIAL PALSY, LEFT SIDE,
Showing secondary spasm same side, and inability to close left eye.

third, *extra-osseous* or *extra-cranial*. The lesions anywhere along the tract indicated in 90 per cent of cases are unilateral. They are all termed peripheral lesions, since they are not in the centre itself. And the paralysis is designated *Bell's palsy*. The extra-osseous form of peripheral disease is frequently excited by exposure to cold and draughts (neuritis), or, as in rare cases, due to trauma. Such a case the author has reported in a girl where a pipe stem entered immediately below the helix of the ear, where it lay buried.

The onset of *symptoms* in the so-called idiopathic cases is, as

a rule, sudden, the patient perhaps waking in the morning and finding the face drawn to the well side. The wrinkles have disappeared from the paralyzed side, the mouth is drawn towards the (Fig. 26) opposite side, and the patient is unable to wrinkle the brow as well on the affected side, saliva dribbles from the mouth; there will be inability to maintain the bolus of food between the teeth, and also some difficulty in deglutition. The patient may also complain of a sense of paræsthesia or numbness on the affected side, but there is rarely anæsthesia, in which case the neuritis has involved the fifth-nerve branches as well. No disturbance of the sense of taste exists. The duration of this type is, on the average, from six weeks to three months, and depending upon the amount of neuritis and destruction of the nerve-fibres will recovery take place completely, perhaps in 50 per cent of the cases fully. The *intra-osseous* portion of the nerve is involved in some of the so-called idiopathic cases of Bell's palsy, but is much more often affected when the patient gives a previous history of otitis media; although this might be the predisposing cause to the extracranial form in rare cases. But with the history of ear disease the disturbance of the sense of taste should be looked for, and will also be frequently found. Taste will be absent or lessened on the anterior two-thirds of the tongue on the affected side in such cases, due to catching of the chorda-tympani nerve as it passes out through the hiatus Fallopii. The testing for deafness should also not be forgotten, and this would be an important point in determining the site of the lesion, since if aerial induction of the tuning-fork is better through the external auditory meatus than when the fork is applied to the temporal bone, the deafness is due to internal ear disease; whereas if the conduction is better through the bone than by the external auditory meatus, the disease causing the deafness and the disturbance of taste is resident in the middle ear. *Intracranial disease* is associated with deafness or vertigo, the latter being a prominent symptom where the semicircular canals are involved, and deafness where the vestibular portion is the original seat of disease. In any case the degeneration of the seventh nerve will be found after the tenth day. *Prognosis* is uncertain.

Treatment.—In any form of Bell's palsy the treatment of the underlying cause is of prime importance, while the prognosis will be good in proportion as this cause is determined and can be successfully *eradicated*; thus, if a meningitis can be controlled early,

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very likely the patient will recover the full use of this cranial motor nerve. Cases due to internal or to middle-ear disease are more serious, since the exciting or predisposing cause is the *difficult condition* to remedy. · Such a case should be referred early to an aurist for proper treatment. Idiopathic cases are best treated by applying a blister back of the ear, keeping the patient quiet, administering a purge and carefully searching for any other cause. The use of potassium iodide should be had early, and in fairly large dosage (from 10 to 15 grains t. i. d.), over a continuous period of two to four weeks, when the dose should be gradually lessened. A smaller dosage should then be continued for a fortnight, and finally the patient put under the tonic influence of strychnine, $\frac{1}{30}$ of a grain t. i. d. The use of galvanism should be instituted by the tenth day or second week after the onset of the disease. (See Electricity, p. 97.) This is the most valuable agent in the successful treatment of Bell's palsy.

Auditory.—Disease of the eighth nerve proper consists of tinnitus aurium, due to irritation of this nerve; and deafness, due to destruction of the nerve-trunk or its centre. Certain forms of paralysis of the seventh nerve, as shown in the preceding paragraphs, may complicate, but they need not be rehearsed here.

Prognosis of eighth-nerve disease depends upon the nature of the disease within the temporal bone or whether the nerve or its centre is involved; in the latter treatment would be of little or no avail, whereas in peripheral trouble, as in middle and external ear disease probably much relief or cure by an experienced aurist may be brought about. A disease in which the eighth nerve is particularly involved is caled *Ménière's disease.* (See Symptomatic Disorders).

Glosso-pharyngeal. — The glosso-pharyngeal nerve supplies motor and sensory fibres to the pharynx, the larynx, back part of the tongue, and sends a branch (Jacobson's nerve) to the middle ear. It is also one of the special sense nerves of taste. (The chorda tympani supplies the anterior two-thirds of the tongue.)

Disease of this nerve would produce, if irritated, an increase of sensibility in the pharynx and hypergeusia, whereas the destruction of the nerve would produce loss of sensibility, with ageusia, on the posterior third of the tongue. Disease of this nerve is usually of central origin, and the prognosis and treatment would depend upon the nature of the ætiological factor of the

primary disease. Usually in disease of the ninth nerve ameliora-
tion is doubtful.

Diagnosis.—This cannot be confused with any other lesions of
any of the other cranial nerves, when one remembers the anatom-
ical distribution of same and its function. *Treatment* is of avail
only as the cause can be got rid of.

Pneumogastric.—This nerve has its origin behind the olivary
body of the oblongata superficially, and has its deep origin in the
floor of the fourth ventricle in close conjunction with the centre
of the glosso-pharyngeal nerve. It supplies the sensory and motor
fibres; through the auricular branch, passing to supply sensation to
the external ear, and a pharyngeal branch supplying motion to the
pharynx. There are also the laryngeal branches, of which the
superior supplies sensation to the larynx and motion to the crico-
thyreoid muscles. The inferior or *recurrent* branch is entirely
motor, and supplies the intrinsic muscles of the larynx, excepting
the crico-thyreoid. Then there are also the cardiac fibres, the
pulmonary and sensory, the œsophageal, and gastric motor fibres.
Disease of this nerve, therefore, produces wide symptomatology,
varying from the absence of sensation to the external ear to paral-
ysis of the pharyngeal muscles, œsophageal, stomach, and laryngeal
muscles, and even paralysis of the heart; so that in the majority of
cases of disease of this centre or nerve itself death results from
heart failure before any other special symptoms could come to the
attention of the physician. Many vague symptoms in the thoracic
cavity are due to a neuritis of the various branches of this nerve.
As a rule, diagnosis is not made until the final symptoms develop
and the patient is carried off by heart failure. The *treatment*
would consist in eliminating causes, such as intoxications or men-
ingitis or polioencephalitis. Strychnine may be of service.

Spinal-accessory.—This nerve has its superficial origin from
behind the olivary body, and its deep origin below that of the
tenth nerve, in the floor of the fourth ventricle, making its exit
through the jugular foramen and supplying motion to the sterno-
mastoid muscle, and it also gives motor branches to the tenth nerve.
Disease of this nerve is usually of an irritative nature, due to func-
tional disturbances of the centre, resulting in spasmodic wry-neck
(Fig. 27) or torticollis. In this affection, which usually has its
onset in early childhood, and is sometimes due to rheumatic taint
or to catching cold, drawing of the head forward and towards the

affected side occurs, the chin being pushed to the opposite side. It is paroxysmal, although there is a persistent tonic contraction of the muscles. If the trapezius is also affected, there will be elevation of the shoulder of the affected side. It is a very chronic disease, and may last for years. No definite pathology has as yet been discovered in the majority of cases. Some are distinctly hysterical, as proved by the fact that mental therapeutics often cure the condition. If it occurs in early childhood, it may deform the chest and neck of the patient so much that the deformity is a very important disability.

FIG. 27.—Showing attitude in Spasmodic Torticollis.

Prognosis.—This is therefore very dubious.

Treatment.—Both medical and surgical measures are to be referred to; medical in the administration of antispasmodics, such as tincture of gelsemium or conium, gtt. v, t. i. d. Surgical, which consists in the excision of the nerve in whole or in part. Some surgeons, as Keen, of Philadelphia, recommend the excision of the cervical-spinal sensory roots. This I have seen cure one patient. Others have been operated on with the reappearance of the deformity. The patient should have a change of environment, and he should live in quietude. Proper gymnastics and chest expansion exercises are especially necessary in young persons. Apparatus is occasionally of service in relieving contractures.

Hypoglossal.—The cortical centres are in the lower part of the central convolutions. This nerve has its superficial origin in the front part of the oblongata and the deep origin in the floor of the fourth ventricle, and passes out through the anterior condyloid foramen. It is entirely motor, supplying the muscles of the

tongue, excepting the palatoglossus, mylo-hyoid, and stylo-hyoid. The *descendens noni* branch communicates with the second and third cervical by the so-called communicans noni nerve. Disease of this nerve consists in paralysis in the majority of cases; irritative lesions seldom, at least, giving symptoms. The cause of destruction of the nerve is either central or peripheral—i. e., is either supranuclear, nuclear, or infranuclear. If central, it is unilateral. If nuclear, it may occur as a symptom in bulbar paralysis, as well as in localized minute apoplexies.

Usually the cause of palsy is neuritis, specific in origin, in which case it is apt to be unilateral, the symptomatology being paralysis of one side of the tongue, fibrillary contractions, and atrophy. The base of the tongue is elevated and the tongue

FIG. 28.—SUPRANUCLEAR PALSY OF THE RIGHT HYPOGLOSSAL NERVE,

Showing tongue deviation to palsied side (Philadelphia Hospital).

protrudes towards the palsied side (Fig. 28). *Diagnosis* of isolated lesion is made by excluding apoplexy or other paralyses due to a very extensive central lesion, and in excluding neuritis from other parts of the body. Atrophy occurs in nuclear or infranuclear lesions.

Treatment.—This depends entirely upon the cause, central lesions and chronic specific disease usually causing permanent disability. Neuritis from traumatic causes would be more apt to be relieved by antirheumatics or by surgical interference. Electricity does not prove of much avail in such a limited area. The use of strychnine is indicated, and tending towards regeneration of the nerve itself. Acute specific neuritis is hopeful if full antiluetic mixed treatment is early administered.

CHAPTER VII

DISEASES OF THE PERIPHERAL NERVES

THE peripheral nervous system consists of twelve pairs of cranial and thirty-one pairs of spinal nerves, together with their root ganglia and terminal sense and special sense organs; also of the sympathetic nervous system. The sympathetic system consists of the intervertebral and the cranial ganglia and the peripheral ganglia. The latter arise during embryonal life from ganglionic cells of the same class as those of the spinal-root ganglia, then later migrate to their position in the sympathetic. According to Minot, therefore, the peripheral nervous system is composed of peripheral motor neurones, peripheral sensory neurones, and peripheral ganglionic neurones.

Origin of Nerves.—Modern studies have shown that the true nature of the nerve-fibre is but a prolongation of the nerve-cell— i. e., of the axis cylinder of the cell. Added to this axis cylinder are supportive structures, also those for insulation when the nerve is complete. Motor nerve-fibres, therefore, come from motor neurones and sensory nerve-fibres from sensory neurones or cells. All peripheral nerve-fibres of motor nerves have in the cord or brain certain motor cells of origin, and these are known as the *nuclei of origin* of these nerves. This is not exactly the same with the sensory nerves. All sensory nerve-fibres take their origin from nerve-cells in the posterior nerve-ganglia, or else in corresponding cranial ganglia, such as the Gasserian, lying upon the cranial nerves. The nerve-cells in these ganglia send off a single process, which divides in T-fashion, the peripheral branch going out to form the sensory fibre, the central branch passing into the cord or brain, and ending in a terminal arborization which surrounds groups of sensory nerve-cells. Hence, these latter sensory nerve-cells are not nuclei of origin, strictly speaking, but are *terminal*

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nuclei. Therefore, there are no nuclei of origin for sensory nerves in the central nervous system, a matter of much importance when we come to consider the anatomy of the cranial nerves.

PATHOLOGY OF THE NERVES

Hyperæmia and anæmia of nerves brings about types of nerve-irritation leading to various forms of neuralgia, of paræsthesia, and of motor irritation or paresis. These conditions are usually, however, secondary, and are but rarely recognized clinically, since it cannot always be determined whether an irritated nerve is anæmic or congested or, indeed, whether the central nervous system may not be mainly at fault.

NEURITIS

This is an inflammation of the nerves. It consists of *interstitial* and *parenchymatous* neuritis. The interstitial neuritis consists of inflammation of the connective-tissue elements of the nerve —the epineurium and endoneurium. The parenchymatous neuritis consists of an inflammatory process, being located in the parenchyma. It is further divided into *local* and *multiple*. In the local form of the disease it is frequently of the interstitial type, whereas in the multiple form it is apt to be of the protoplasm of the nerve. It is also divided into acute, subacute, and chronic.

Symptoms of *acute neuritis* are pain along the course of the nerve-trunk, tenderness, moderate fever, and lessening of the reflex in the parts involved, with perhaps hypæsthesia or anæsthesias, depending upon the extent of the lesion. After the first few days the tenderness may largely subside, the sense of paræsthesia lessen, and the condition settle into a more regular course, somewhat below the severity of the onset—i. e., with lessened spontaneous pain and tenderness. If localized, an active case may terminate within six weeks, or, if general neuritis, the acute may be designated to those cases continuing a greater length of time. The reaction of degeneration may be found as early as the seventh day of the disease.

Causes.—Traumatism, poisons from within and without, infectious diseases, etc.

Diagnosis would be only between myalgia and neuralgia, in which latter case there is no tenderness on pressure, but rather an

alleviation of the pain; nor does elevation of temperature, local or general, exist in neuralgia. In myalgia the muscle pain would be relieved by firm pressure, while in myositis the belly of the muscle would be painful to the grasp, but no nerve-trunk tenderness would exist.

Prognosis is guarded and depends upon general state of health, as well as upon the severity and persistence of the cause.

Subacute neuritis consists of the disease after it has run longer than the average duration of six weeks, or when the nature of the infection has been very slight from the beginning, and consequently the onset gradual. In the subacute type we have a modification of all the symptoms; pain and tenderness are much less severe, weakness of extremities and muscles less pronounced, both on account of the fact that the nerve structure is not disabled functionally and that there is less pain at the time of attempted muscular acts. Reaction of degeneration may be pronounced. The fact that a case is subacute immediately makes the prognosis more guarded, since the name itself implies a chronicity of the condition or a predisposition to the inflammation of the nerves in a particular case.

Diagnosis.—It is likely to be mistaken for neuralgia, but may be confused with chronic rheumatism, the difference between the two being, of course, the lack of localized tenderness along nerve-trunks in rheumatic affections. If a rheumatic neuritis exists, there will also be found other evidences of the disease (rheumatism), such as fever, joint or cardiac involvement, with excess of urates in the urine.

Chronic neuritis is that form which persists longer than three months, and is, as a rule, the sequel of the acute or subacute disease. The *symptoms* are much more modified than either of the two preceding varieties, although localized atrophy of muscles may be much more in evidence; also, reaction of degeneration will be more distinctly shown, while the pain and tenderness and anæsthetic areas along the course of the nerve-trunks will be very likely slight, but on the other hand, from the prolonged disability and muscular atrophy, palsy is much more pronounced, and the reflexes may be entirely abolished, although in some of the chronic interstitial types of mild grade I have even seen *increase* of the deep reflexes, as the knee-jerks. When the latter occurs, it can be explained by the irritation of the nerve-fibres through inter-

stitial connective-tissue overgrowth, which re-enforces the reflex impulse.

Diagnosis.—The difficulty lies between it and chronic neuritis of rheumatic type. The symptom pain may be confused with the pain of cord disease, as in tabes. In tabes we would have other evidence of spinal-cord lesion. There would be nó tenderness at all, while anæsthesia or other sensory changes would not vary much from week to week in cord lesions as it may in neuritis. As between rheumatism and neuritis, we have given points above which need not be repeated here.

Prognosis.—This depends on the history of alcoholism or the use of other poisons, upon the vulnerability of the patient, upon the diathetic tendency of the individual towards rheumatism, etc., and upon the locality in which he resides, since a low, damp country would be particularly bad for this malady. As a rule, patients will recover if proper treatment can be administered early.

Treatment.—The treatment of *acute* neuritis, whether local or multiple, has certain fundamental principles to be observed. In the first place, rest of the part involved as nearly absolute as possible, is a desideratum. The relief of pain is due largely to this measure as well as to cure of the inflammation itself, which is thus favoured by absolute immobility. A splint is often a useful measure for producing this quietude. If the case is one of multiple neuritis, the patient should be placed in bed; in the acute form, and in many cases of subacute and chronic types cure will be had much more quickly if this measure is adopted earlier than is at present the general practice. For relief of pain in the acute form, local applications of lead water and laudanum (25 per cent) are also of value; the arm should be protected by means of cotton or heavy wool. Particularly if near a joint, the extremity should be splinted. Among drugs, the analgesics will do much to control pain, such as phenacetine, antipyrine, etc., in 5- to 10-grain doses, as required. Finally, hypodermics of morphine may have to be given to control this symptom. The use of sodium salicylate is commended, particularly in those cases of rheumatic origin. Also the employment of potassium iodide as an alterative is of value. In chronic cases, with reaction of degeneration, the use of massage and galvanic electricity on alternate days are valuable adjuncts after the acute pain and tenderness have subsided. In acute cases the diet should be limited, but in chronic cases, where the patient has

been run down, the use of nutrients is of *par excellence*, such as meats, vegetables, milk, etc., and even cod-liver oil, although in cases of rheumatic origin the use of proteids should be guarded, according to the idiosyncrasy of the individual. The counter-irritation over the course of a nerve, by means of blistering, or the actual cautery after freezing is frequently of value. The use of strong tincture of iodine is another irritant I have seen do good service.

Multiple neuritis as well as other forms may be idiopathic, or its ætiology may be from sources already mentioned under the general head of neuritis. A better name for the idiopathic form would be essential, since this does not imply that there is not a cause, even though not discovered. The more frequent causes, however, are alcohol and lead and exposure or infectious diseases, as typhoid (Fig. 29). This type of the disease pathologically is usually of the parenchymatous variety.

Special Forms of Multiple Neuritis.—In the *alcoholic* form of multiple neuritis there is early toe-drop, some pain along the course of the nerves of the legs and arms, loss of knee-jerks, which may indeed be absent before the patient complains of weakness or pain in the extremities. There is also considerable wasting of muscles, with reaction of degeneration coming on early in the palsy. The patient presents the gait which is styled the "steppage gait," due to the weakness of the anterior group of muscles and sequent toe-drop. There will also be found to be extreme gastric disorder, as a rule; a gastric catarrh, and pains in the abdominal viscera, due to the alcohol irritating the mucous membrane of the stomach, with subsequent inflammatory condition of the entire gastro-enteric mucous membrane. There may or may not be marked wrist-drop in alcoholic neuritis. The heart may be affected, the patient suffering from palpitation and arhythmia, and in some cases death may result from involvement of the vagi nerves or centres. Palsy of the extra-ocular muscles may produce a strabismus, diplopia. In some cases atrophy of the optic nerve itself; and blindness may follow due to the extension of the inflammation of the optic nerves. The nails often become brittle and transversely ridged, caused by lack of growth at special epochs in the course of the malady. Frequently the patient becomes the subject of a progressive muscular atrophy, the result of degeneration of the nerve protoplasm. In alcoholic neuritis pain

is not a prominent symptom (but it is very pronounced in the so-called idiopathic cases). The duration of the disease may be from six months to several years, the patient recovering at the end of this time in the majority of cases under proper treatment.

In *lead multiple neuritis* the symptoms are somewhat different from those due to alcohol, inasmuch as wrist-drop is a very distinctive and early symptom, toe-drop being rather a secondary or late phenomenon. In lead neuritis, the onset is more rapid, and frequently preceded by gastro-intestinal disturbances, such as tormina, vomiting, or other evidences of lead-poisoning. Another point is the fact of the blue lines being about the gums. This is due to reaction of the lead upon the saliva producing lead sulphocyanide. The blue line is always at the junction of the teeth and gum, but is upon the latter. The blue line, however, does not exist when there are no teeth present, it must be remembered, since the

FIG. 29.—BED-RIDDEN CASE OF MULTIPLE NEURITIS (post-typhoid infection).
Showing toe- and wrist-drop.

tartar is not then present to produce the characteristic chemical reaction.

Diphtheritic Multiple Neuritis.—Another variety of multiple neuritis fortunate enough to be given a special place, and yet many times overlooked by the general practitioner, is the diphtheritic form, which may or may not be associated with the *post-diphtheritic pharyngeal paralysis*. This I have recently seen in the case of a doctor who had been a most healthy specimen of manhood, and who four weeks after the onset of severe toxic diphtheria developed neuritis. In him the palsy of the throat was

.very slight, but the universal involvement of nerves seemed to show the very profound intoxication of the nervous system. In diphtheritic neuritis, the history of diphtheria, or, if the case had not been closely observed, simply the story of a sore throat, will be the deciding factor, plus the inability early to swallow well or the incapacity to lift the soft palate as in saying " Ah ! "

Arsenical neuritis is also a type of multiple neuritis, and is frequently caused by prolonged medication in the use of Fowler's solution or of arsenious acid, either due to the patient taking the drug for cosmetic effect, or perhaps in the treatment of chorea.

Diagnosis of arsenical poisoning resulting in neuritis cannot be positively made without a previous history of poisoning, since there are not sufficient distinctive features to differentiate this. The physician should always look to the history of the patient sleeping in a room where arsenic has been deposited in wall paper prepared by the arsenical process, not a few cases having been reported through investigations by Shattuck, of Boston.

BERI-BERI OR KAKKE

This is a type of multiple neuritis, particularly endemic in the Philippine Islands. It is probably infectious. Some authors claim it is caused by CO_2 poisoning, others that it is due to diet of fish and rice containing fungi. It is associated with œdema, effusion into the serous cavities, such as the pericardium, the peritonæum, or into the ventricles of the brain. It is marked by paralytic and atrophic disorders, anæsthesia, and lightning-like pains. Death is the result of the effusions rather than of the neuritis proper, or else to early involvement of the phrenic or vagi nerves. Mental disturbances are frequent.

Prognosis.—This is absolutely bad.

Treatment of multiple neuritis does not differ in any *chronic* type except in so far as separate causes are determined—namely, alcohol, lead, diphtheria, or arsenic. Treatment of the resulting neuritis would, at least, be the same in any case.

In the types mentioned there is the possibility for aid through early elimination of the poison. The use of potassium iodide is a valuable measure as an eliminant in all cases due to poisoning by drugs. *The source of infection should be cut off.* Besides, the patient should take large draughts of water. The persistent use of general massage daily, fresh air and sunshine, plus the use of

cod-liver oil and hypophosphites, living in a dry climate away from prevailing high winds, will insure success if carried out over many months in cases where chronic invalidism has occurred from the lack of persistent endeavour in these directions by either the patient or physician. Strychnine should be given in large doses in cases of chronic neuritis—$\frac{1}{30}$ to $\frac{1}{10}$ being administered thrice daily. The use of galvanism is also a valuable measure where there is degeneration of nerve tissue. In this case the negative pole should be placed over the spine and the positive over the atrophied muscles, a constant current being applied to all the palsied muscles for several minutes three or four times a week. Massage is of great value when administered daily. Alcoholic cases must be rigidly guarded against indulgence in the drug, since a small amount of alcohol will precipitate inflammation. Auto-intoxication from intestinal fermentation should be prevented by restricted diet and the use of salol in moderate doses (2 grains t. i. d.).

Erythromelalgia (see frontispiece) is a variety of multiple neuritis affecting the dorsal and plantar nerves of the foot. It was first described by Weir Mitchell, who gave it the name of a vasomotor neurosis. We have had the pleasure of studying two of Mitchell's cases. One was due to a crush injury caused by the falling of a heavy stone upon the foot. The case cured through stretching of the plantar and by excision of the cutaneous nerves. The other case died of a complication of gangrene of the foot following infection after the operation of excision and stretching of the internal saphenous and musculo-spinal nerves, and of stretching of the posterior tibial nerve. Histological examination showed neuritis, thickening of the middle coat of the vessels of the amputated (affected) foot. This obtained in the smallest arterioles, while the larger vessels were also calcareous. This type of neuritis has particular characteristics. When the affected foot is pendant it becomes intensely red; a severe burning pain is set up, in which the local temperature rises to as much as $\frac{4}{5}$ ° C. higher than when the foot is held horizontally. This is the opposite of what occurs in a normal extremity, as Mitchell points out in his Clinical Lessons on Nervous Diseases, pp. 202, 203. Walking frequently is so painful as to be almost impossible. Excess of perspiration may occur when the foot is pendant or is paroxysmally painful. Heat aggravates the pain also, while cold relieves it. Local

ulceration from profound trophic disturbance follows in bad cases.

Prognosis.—Usually chronic and persistent. Spontaneous cures have occurred.

Treatment consists in rest in bed, local applications of cold, protection of the foot by cotton or splints loosely applied. Galvanic electricity, the positive pole being placed over the affected part, together with effleurage, may afford relief. Stretching and excision of nerves are measures to be adopted where relief fails from other forms of treatment. The drug habit should be guarded against.

Recurrent multiple neuritis is a type described by Sherwood, Ross, Osler, and others. Certain individuals are susceptible to recurrent attacks from definite poisons, particularly from alcohol and lead. Since it is not necessary for the intoxicant to be again taken to produce recurrence of the neuritis, there must be induced in such patients a susceptibility. Each recurring attack is likely to be more severe than the former.

Treatment is the same as for other forms of multiple neuritis. It is especially dangerous for these patients to be exposed to the poison originally causing the disease.

Leprous Neuritis.—The leprous bacilli may cause neuritis. Proliferation of the nerve-trunks occurs, nodules are formed, and finally the bacilli may disappear. According to Martius and Sonza, the spinal cord may become invaded by the bacilli and cavities form, especially in the posterior horns and in the gray commissure. Sensory symptoms, particularly of anæsthesia, occur, especially in large patches on the face, hands, forearms, feet, and legs. Through spreading, a very large area may thus become anæsthetic. The small muscles of the hand may be wasted, and even the bones may become atrophic. There may occur deformities of the hands and feet due to atrophy of the parts, followed by contractures. The toes, fingers, hands, and feet may even be cast off, as in dry gangrene. The neuritis may last many years. Syringomyelia of the Morvan type may be simulated in some cases through existent dissociation of cutaneous sensation, very likely caused by the disease in the gray cord as noted above.

After-treatment of Neuritis (continued).—In the toxæmic state following the acute infections, large doses of iron, as of the tincture of the chloride, are of great value. If malaria is present, quinine and arsenic are important remedies. The cachexias that

may exist as predisposing causes should be sought for and, if possible, remedied. In cancer, tuberculosis, or in pernicious anæmia, the measures that cause general improvement will aid in neuritis. In bad cases it may be necessary to pass food through the stomach-tube. If the heart is at all enfeebled, such cases should be watched most anxiously and quietude enjoined. At times a brachial neuritis will thus end in death suddenly by extension of the neuritis through some anomalous branch connected more directly with the vagi or phrenic nerves, or else it may be a reflex inhibition of the heart that occurs.

Contractures and posture deformities must be very carefully guarded against, especially in alcoholic cases. The weight even of the bed-clothing must be taken off the toes by means of " hoops " or other improvised apparatus. At times the use of splints, where there is not extreme sensitiveness, will aid much in preventing contractures; as about the ankles. Passive movements in full extension, and especially in full flexion of the ankle, should be used several times a day in the alcoholic type. Heat applied will at times do good in relieving pain. We have seen cold occasionally do good, but for some unknown reason it invariably aggravates any form of neuritis of the upper extremities, though it may do good, as indicated in sciatic neuritis.

Hot baths, Turkish and Russian baths, or needle douches, as advised by Charcot, may all be employed late in the course of the disease. As with the coal-tar derivatives, the use of morphine or cocaine should be very guardedly employed. The bromides and chloral combined allay cerebral irritability and produce sleep more safely than any other drugs. Static electricity is of value to awaken the circulation and aid metabolism. This and the faradic brush will frequently greatly benefit the cutaneous anæsthesias. The patient must be encouraged to walk as soon as possible, since the muscles gain power by exercise. Tenotomy may be required where contractions have occurred. The use of deep kneading about indurated joints caused by disuse and the inflammatory exudate, is of great value, especially if associated with a hot bath (180° F.) in an alkaline solution (such as of soap), the Turkish bath, or Scotch douche.

SCIATIC NEURITIS

This has in the past been confused with neuralgia, or sciatica, but where tenderness is present neuritis must exist; and as a rule,

sciatic disease is neuritic in origin.   Sciatic neuritis is a very important disease clinically.

Ætiology.—The causes are like in other instances of neuritis—*general* and *local*.   It is more frequent after twenty-one years of age.   Gibron (London Lancet, 1893) says it is eight times as common in males as in females, and this is no doubt due to greater exposure of men.   Hence, exposure to cold and dampness is one of the most frequent causes.   It is common in puddlers, miners, stokers, and cabmen; neurotic, rheumatic, and gouty subjects are very liable to it.   Lead-poisoning favours it.   Diabetes, syphilis, typhoid fever, la grippe, and malaria predispose to its development.   It may follow operations for lithotomy, childbirth, or from pelvic disease in women, or aneurysmal pressure.   Bony thickening of the sciatic notch or gumma, in rare instances, are causes of sciatic neuritis.

Lumbago may precede or complicate (extension by contiguity). *Compression* of the nerve may be a cause, as in sitting a long time on a hard bench, as in shoemakers; or it may be a direct contusion of the nerve, as from a blow.   Excessive fatigue of the legs may precipitate an attack, as in prolonged use of the sewing-machine.   The pressure of varicose veins may produce it.

Spinal disease and new growths in the spinal canal may cause sciatic neuritis by first affecting the nerve-roots, the vertebral canal, or at the cauda equina.

Symptoms.—Pain and tenderness along the course of the nerve are the principal symptoms.   While both of these signs may extend throughout the sciatic distribution, they are more intense in the upper half of the thigh near the sciatic notch.   The tenderness is often extreme, and the patient may be able by this to trace the nerve down to the popliteal space.   Below the knee the pain is likely to follow the external popliteal.   The pain is more or less constant, and is very wearing upon the patient, especially since exacerbations occur mostly at night, keeping the patient awake, when physiological resistance of the nervous system is lessened. Flexing the thigh well down upon the abdomen, or walking, creates added pain very quickly.   Partial flexion of thigh and knee, however, with extension of ankle (a characteristic position when the patient lies abed), will relieve pain through relaxation.   The characteristic gait of semiflexion and limp is due to the same semiflexion.   If it be a chronic case, lateral curvature of the spine may

develop, the convexity being towards the affected side. This sco-
liosis, being muscular, disappears on recovery. A lumbar con-
cave curve and dorsal convex curve of the spine may develop in
those rarer cases, with spasm on the affected side due to neuritic
extension to the anterior crural nerve, the sacral plexus, or lumbar
cord. This is termed *homologous sciatic scoliosis,* and is likely
to become permanent, as the sciatic association symptoms are
chronic and contractures develop which fix the peculiar deformity.

Particular points of exquisite tenderness in sciatic neuritis are
designated *tender points* of Valleix, such as the *gluteal point* over
the sciatic notch, the *trochanteric point* over the great trochanter,
the *popliteal point* at the division of the nerve, and the *fibular
point,* where the external popliteal winds around the head of the
fibula. There may also be a *tender point* on the dorsum of the
foot. In addition, in some cases there may be an *iliac point* about
the middle of the crest of the ilium, *lumbar points* just above the
sacrum, or a *patellar point* over the patella; *points* behind the
malleoli, in the *calf,* or *plantar points* in the sole of the foot.

It is at these points that the patient complains of *paroxysms
of pain* occurring in the course of the disease. This pain may
be boring, burning, tearing, or lancinating in character, which
tortures the patient to distraction. Using the limb or " catching
cold " will frequently at once precipitate an attack. At times
the sudden attacks of pain are spontaneous and periodic, as in
malaria. *Cutaneous sensibility* is often disturbed. This may be
of hyperæsthesia in the sciatic distribution followed by anæsthesia,
or the sensitiveness may be confined to the areas of the painful
points just given. Paræsthesias, such as feelings of formication,
fulness, pricking, or of heat or cold, may be present.

Motor symptoms, as a rule, only appear in the grave cases.
Cramps, violent tremor or spasm of calf muscles may be present
and increase the pain. *The knee-jerk is rarely exaggerated*; usu-
ally it is much diminished, and the *heel-jerk* may be absent. If
there is atrophy of muscles fibrillary twitching is often found, and
there is then palsy or paralysis. In the worst case a severe spasm
involving the entire limb may be produced, at times creating the
most profound distress. A slight draught of air or even the sud-
den touch of clothing may bring on this spasm, so that the limb
must be well protected from both. *Vaso-motor* disturbance may
cause flushing or, in severer cases, lividity of the limb, or even

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œdema. Usually the sweat is reduced. It may be increased. Erythema, acne, perforating ulcer of foot, herpetiform eruptions, or scaliness may all be found in some cases. Reaction of degeneration may be elicited in the wasted muscles. In thin people the swollen nerve may sometimes be easily palpated. Excess of fat may mask the muscular wasting.

Double sciatic neuritis is rare and is usually the result of pelvic or spinal disease, which makes the diagnosis easy.

Pathology.—Perineuritis and inflammation of the adventitia are present. The nerve parenchyma suffers secondarily. The nerve is red and tumefied (œdematous). It is therefore distinctly interstitial neuritis primarily.

Diagnosis.—This must be made from neuralgia or muscular rheumatism. In the former the pain is fleeting, and there is no nerve-trunk tenderness. In muscular rheumatism the pain is diffuse, and the muscles about are sore to touch. Hip disease is determined by the joint tenderness and fixation and pain in the region of the obturator nerve. Hysteria may be simulated, but is distinguished by the fleeting character of the pain and the emotionalism or other stigmata. The pelvic organs and spine should always be looked to as a possible source of the inflammation of the nerve. If the disease is bilateral it has pretty positively come from within the pelvis, as from a growth, or as indicated above. History of syphilis, malaria, or of exposure, often tells all. Injection of a local anæsthetic may stop pain for a time when it has begun or is entirely located in the nerve, not above the notch.

Prognosis.—In uncomplicated sciatic neuritis the ultimate result is good. If due to spinal disease, the prognosis is to be guarded, since cure of the original trouble is doubtful. The average case will last several months, but if the part is used the neuritis may last for months or even years. Relapses are common. If the limb is wasted it may take a year to recover full use of the extremity after the pain has subsided.

Treatment of Sciatic Neuritis.—This is allied to that of treatment of any local neuritis. Special measures consist of absolute rest of the limb, the patient being put to bed. Or a long, well-padded splint can be applied from axilla to external malleolus, and the heel should be protected by raising it from the bed by a pad over the tendo-Achillis. The limb should be thus held in a partially flexed position, first having applied a flannel bandage from foot to

hip for protection and warmth. Diathetic states, as of rheumatism or gout, must be treated if present. Examination of the urine is very important as to determination of this. Counter-irritation by means of strong tincture of iodine thrice repeated, or the use of a blister, or of the actual cautery of Paquelin, are all of great service. Extremes of heat or cold may do good in individual cases. Thus, ice-bags or hot poultices kept applied along the course of the nerve may give great relief to suffering. I have seen sodium salicylate, gr. x, every three hours in peppermint water, do good in acute cases. Potassium iodide in chronic cases or injection of cocaine or atropine down near the nerve may be of much service. But other fluids injected into the tissues the writer has not seen of any service whatsoever, nor has puncturing the nerve-trunk been productive of any relief, and there is some danger of re-exciting more active inflammation. Pressure upon the nerve, as recommended by Negro, repeating the bloodless procedure some six times every third day, may aid resolution in chronic cases with sheath thickening. Massage likewise is only valuable in chronic cases. Electricity is of most value when used twice or thrice weekly, the positive pole being applied over nerve-trunk and calf (10 to 15 milliampères). Faradization may be of service late in the case fo develop atrophied muscles. Nerve-stretching by the bloodless measure of thigh flexion, the knee being extended, or by open incision and lifting the limb from the table by a dull hook applied around the nerve-trunk, are required in stubborn cases. Tumours near or on an inflamed nerve should be excised. Morphine hypodermically may be required in bad cases for the pain. It seems to produce a good effect upon inflammatory tissue. Alkaline mineral waters, as the Buffalo lithia or Londonderry lithia, or a course of alkaline baths, as at Virginia Hot Springs or at Baden abroad, may be of great value.

TUMOURS OF NERVES

A. *Hyperplasia.*

B. *True neuroma; single benign.*

C. *False neuroma; multiple benign, or malignant.*

A. *Hyperplasia* or *hypertrophy* of nerve-trunks is rare. It is usually due, therefore, to increase of the interstitial connective tissue. At times there is an increase of fibres and thickening of the myelin sheath.

B. *True neuromata* are also very rare, and occur most exclusively on spinal nerves. In some instances there is an increase in medullary fibres, in others only an increase of non-medullated fibres—i. e., only the axis cylinders and neurolemma increase. These tumours may occur single or multiple. *Multiple neuromata* are generally, however, neurofibromata. Nerve-fibres massed in hyperplastic connective tissue form plexiform neurofibromata.

True neuromata are usually quite small, measuring several centimetres in diameter only, but they *may* be much smaller or larger. They are usually few in number, though there may be a local multiplicity of these true neuromata. Gowers has shown that in one case 1,000 were present, and this number may be exceeded in rare instances.

C. *False Neuromata.*—This term is applied to nerve tumours intermixed with fibroma, myxoma, glioma, sarcoma, carcinoma, or syphiloma, although the first is the most common variety. Gumma has only been found in the false neuroma on the intracranial or intraspinal nerves. Scirrhous or medullary (rarely the colloid type) carcinoma are the varieties found in combination. Leprous neuritis sometimes forms neuro-fibromatous swellings.

Malignant Neuromata.—Some 30 cases of very malignant neuromata have been recorded in the literature. Hereditary influence and trauma are the ætiologic factors. The great nerve-trunks are most frequently affected, such as the medium or sciatic. The tumours start from the perineurium, and are at first spindle-shaped ; they may grow very large. Sarcomatous cells are the most frequent, but myxomatous cells may be found in them, or fibromatous tissue, or the tumours may be *mixed*.

Tubercula dolorosa are simply false neuromata situated superficially at the ends of sensory nerves.

Ætiology.—A. *Heredity* generally tends to cause the true multiple or plexiform neuromata. B. *Diathetic*—e. g., tuberculous—influences which produce various forms of tumour formations—sarcoma, carcinoma, etc. C. *Injuries* or surgical operations, which cause especially the fibroneuromata, as the *amputation neuroma,* forming often the so-called *irritable* stump.

Neuromata may be congenital, and are then usually of the plexiform type. Multiple neuromata may develop in early life. Men are more liable to neuromata than women.

Symptoms.—Neuromata may not produce symptoms, but the

most frequent evidences of their presence, besides physical exam-
ination, are pain and tenderness. Pressure above the tumour over
the affected nerve may at times relieve the exacerbations of pain
probably by obtunding sensation or cutting off the blood supply
temporarily. Paræsthesia, anæsthesia, paralysis, and reflex spasm
may be found. I had a case recently of false neuroma upon the
median nerve where spasm of the biceps muscle was most annoy-
ing. Some forms of persistent headache are due to multiple neu-
romata of the sensory fifth. Single neuromata cause symptoms
more frequently than multiple or plexiform because they are
points of irritation, as a rule, near the periphery of the nerve;
and connective tissue contracting in them also aggravates pain,
the multiple type being purely nerve tissue. Neuroma of the
splanchnic or vagi nerve may cause most serious symptoms (sudden
unaccountable deaths are at times due to neuroma of the vagus).

Multiple (true) neuromata may last for years and cause no
serious symptoms or inconvenience. Malignant neuromata cause
such symptoms as always result from irritation or compression
of a nerve.

Diagnosis.—The diagnosis is only positively made when palpa-
tion reveals the tumour. In other cases diagnosis is made by ex-
clusion. Lipoma over the site of a nerve is the most easily con-
founded with neuroma. (The liability of lipoma to appear on any
part of the body should always make this innocent tumour thought
of in diagnosis of any growth.) True neuromata are often multi-
ple; the false are usually single. *Idiocy, heredity, neuropathic*
constitution would always favour the disease being true neuroma.

Treatment is essentially surgical. Internal medication and
applications are valueless save as the former is towards general
building up of the constitution to the invasion of disease. Strong
galvanic currents (descending), mercury, and the iodides in large
doses may assist in the resorption of exudate about such a growth.
Excision is the proper treatment if pain or paralysis are prom-
inent symptoms. The growth may return.

CHAPTER VIII

DISEASES OF OTHER SPINAL NERVES

A SINGLE spinal nerve may be injured by disease or trauma, or several nerves may be injured by the same local conditions. The principal disturbances resulting are those of sensations, or of motor or trophic disorders which manifest themselves within the distribution of the nerves involved. (See Fig. 8.)

The most usual lesions are new growths, division, degeneration, neuritis (see Chapter VII). When the lesion is *irritative,* the symptoms are of hyperæsthesia, pain, spasms, tremor, spasticity, and rarely of hypertrophy. When the lesion is *destructive* from the first or has passed on to the destructive stage, the symptoms produced are such as anæsthesia, palsy or paralysis, of dystrophy, atrophy, or wasting. The above two groups of symptoms are not infrequently blended, the result of partial involvement of a *special* nerve or of the sensory-motor fibres being affected to a different degree in a *mixed* nerve.

Cervical Plexus.—The phrenic nerve arising from the third, fourth, and fifth cervical nerves may be impaired in function by penetrating wounds of the neck, as by a pistol shot, or by new growths, or aneurysm causing pressure upon it. Disease of the cervical vertebræ, or disease of the spinal meninges, may finally affect the nerve-roots or spinal nuclei. Inflammation, *primary* or *secondary,* from contiguity with seats of inflammation, may be a cause.

Sensory disturbance in disease of the phrenic nerves may simulate the pain of intercostal neuralgia or of rheumatism; but its association with the *motor symptoms* will clear this up. The latter will consist of inactivity of the diaphragm, unilateral or bilateral, as shown by failure of the abdominal walls to advance with deep inspiration. If the lesion is of both phrenic nerves the breathing is distinctly of feminine or costal type; the dyspnœa is extreme.

The suprascapular nerve, which arises from the fourth, fifth, and sixth cervical nerves, may be affected singly in dislocations of

the shoulder. If so, the supraspinati and infraspinati muscles become atrophied, the scapula being prominent. Also, the external rotation of the arm is impeded, with general weakness of the member due to lack of balance at its fulcrum, the shoulder-joint. Writing is very difficult for the reason given. There may be an *anæsthetic* area over the outer aspect of the scapula and posterior portion of the deltoid region.

· **The long or posterior thoracic nerve,** branching from the fifth and sixth cervical nerves and supplying the serratus muscle, is quite frequently exposed to mechanical pressure, as from heavy weights carried upon the shoulder by expressmen, etc.; or it may be compressed by muscular efforts exerted by overhead workers, as plasterers, etc.; or by swinging the arm constantly, as in chopping, mowing, or tailoring. For all these reasons men in active early life are more frequently affected, and usually on the right side, because the majority are " right-handed." The nerve may also be affected along with others in the course of spinal disease.

Symptoms are mostly *motor*, since it is almost a pure motor nerve. These consist of impairment of expansion of the chest on the side affected, and of diminished movement of the upper extremity, as a result of the non-fixation of the scapula. The intrinsic muscles of the arm itself are not weakened. The deformity from paralysis of the *serratus* produces a peculiarly characteristic disfigurement. Placing the arm forward causes the posterior border of the scapula to widely wing out from the chest, so that a deep recess is formed behind the shoulder-blade. The upper portion of the scapula also moves outward, and the lower angle approaches the spine. *Sensory* phenomena are but slight, and consist of pain in the neck and shoulder in case neuritis coexists.

Prognosis is less favourable than in injury to other spinal nerves, even though the disease affecting it is a simple pressure neuritis. This may be due to the fact that pressure is usually made along a much greater extent than in the case of any other nerves that may be so injured.

The *treatment* of palsy of the *phrenic* nerve will be entirely confined to treating the cause, since the nerve is inaccessible to mechanical stimulation, and this is largely so with palsy of the suprascapular or the *long thoracic*; but galvanism can be employed to advantage in some cases by placing the negative pole over the digastric region and the positive down the border of the scap-

ula on the affected side.   Strychnine, gr. $\frac{1}{60}$ to $\frac{1}{10}$ three times a day, may prove of value.   Massage is of most service.

CERVICO-OCCIPITAL NEURALGIA

The *symptoms* of neuralgia of the first four cervical nerves, especially of the occipital branch, are caused by exposure to draughts, catching cold, rheumatism, etc.   Pain here may also be due to caries of the cervical vertebræ.   It may be associated with neuralgia of the fifth nerve and with torticollis.   The pain is usually located in the occipital and at times in the posterior parietal region.   It may be bilateral.   There may exist tenderness of the scalp.   Tender spots are found (*a*) in the triangle between the trapezius and sterno-cleido-mastoid muscles; (*b*) at the angle of the great occipital between the mastoid process and the spine; (*c*) above the parietal eminence.

Prognosis is usually good excepting where the disease comes on late in life.

Treatment consists in counter-irritation to the neck and occiput by means of strong tincture of iodine, the fly-blister, or the actual cautery.   Galvanism with the positive pole of a galvanic current placed over the three tender points indicated above is of value.   A hypodermic injection of morphine, gr. $\frac{1}{4}$ to gr. $\frac{1}{2}$, guarded by atropine, gr. $\frac{1}{150}$, may be required *during* an exacerbation of pain.   Quinine in full doses or sodium salicylate pushed to the physiological limit are of value.   Arsenious acid is a desirable alterative in these cases, especially where anæmia complicates, when Blaud's pill, gr. v, t. i. d., should be added.

The Circumflex Nerve.—This nerve arises from the fifth, sixth, and seventh cervical nerves, and descends in the posterior cord of the brachial plexus.   It then branches off, to pass outward under the deltoid muscle, and winding around the neck of the humerus is distributed to the teres minor and deltoid muscles, also supplying the shoulder-joint with trophic influence.   It also supplies sensation to a chevran-shaped area over the lower two thirds of the deltoid.   From its exposed position it is often injured in shoulder dislocations, in arthritis, or by falls or blows upon the shoulder; or by crutch pressure.

Symptoms of paralysis of the circumflex nerve consist of inability to abduct the arm from the body due to deltoid paralysis,

while the palsy of the teres minor is insignificant. Soon the del-
toid wastes and the shoulder becomes pointed and prominent, with
the shoulder-joint relaxed from sequent dragging upon its liga-
ments. The head of the humerus can be felt very readily. Nutri-
tion of the joint failing, it becomes the seat of neuro-arthritis and
partial ankylosis.   This is ascertained by noting synchronous
movement of the scapula with arm motion.   An initial arthritis
may likewise spread by continuity to the circumflex nerve and
injure the deltoid. Anæsthesia over the skin distribution of the
deltoid, already described, is usually present in circumflex paral-
ysis.

Prognosis is guardedly favourable in cases not due to frac-
ture.

Treatment consists of blistering, sodium salicylate in rheumatic
cases, and after the tenth day galvanism, massage, and move-
ments, kept up for months.

Paralysis of the infrascapular nerve, which arises from the
fifth and sixth cervical roots, consists of palsy of the *supraspina-
tus* and *infraspinatus* muscles, which it supplies; but isolated
paralysis of these muscles is rare, palsy of this nerve usually being
associated with deltoid palsy from circumflex-nerve disease due to
dislocation of shoulder-joint, etc. Paralysis of these two muscles
alone, however, causes loss of power of outward rotation of the
humerus, shown in loss of power of carrying the hand from left to
right, as in writing.

Prognosis is also guarded, since it is difficult to determine the
extent of injury.

Treatment is exactly similar to that of circumflex palsy, save
that massage and electricity are applied directly over the muscles
involved; so that treatment is more available at least.

The musculo-spiral nerve arises from the posterior brachial
cord, and winds around the humerus in the musculo-spiral groove
between the two heads of the *triceps* muscle, where it can be sub-
jected to muscular pressure, external violence, or pressure.   It is
the most frequently diseased nerve in the body, and at the same
time is the one in which disease is best in its *prognosis.*

This nerve supplies all the extensors of the elbow, wrist, and
fingers; also both supinators, and through its radial branch the
skin on the dorsal surface of the thumb and two radial fingers,
and the posterior radial border of the hand.   It furnishes as well

trophic-joint filaments to the carpus and hand. High up it also supplies cutaneous branches to the skin in an area extending from the wrist in a narrow but widening strip up the forearm, and over the outer aspect of the arm as high as the insertion of the deltoid muscle. These latter branches are but seldom involved in palsy of the circumflex.

Causes of disease of the circumflex nerve are legion. It may be due to lead-poisoning, when the posterior interosseous branch ("nerve of lead palsy") is most affected, wrist-drop being the only prominent symptom in such cases. In palsy of the circumflex from drug-poisoning, it is also interesting to note that the supinator longus is not affected. From its exposed position in the axilla crutch pressure frequently causes pressure palsy; or it may here be due to dislocation of the head of the humerus. Lower down the nerve is liable to suffer from blows upon it, by being caught in callus or fractures, or by a cord being tied about the arm, or even by direct action of the triceps catching the nerve in its grasp, as in extreme muscular exertion. By far the most common cause is from pressure by the patient lying on his arm, or when it hangs over a chair, he being asleep, in alcoholic poisoning. The reason for this is that the alcohol benumbs sensibility and the patient is not awakened by the paræsthesia that would occur under normal circumstances. Of course, such pressure may occur under any form of narcotism. The surgeon must be careful not to have *pressure* upon this nerve, in operations, while the patient is anæsthetized. Hanging pendant when under an anæsthetic may also produce pressure palsy from the *pull* made on the nerve. Finally, "catching cold" may be the cause of neuritis and palsy of the circumflex.

Symptoms.—*Sensory* symptoms are slight and at times not present when the motor fibres of the nerve are almost completely functionless. If present, it would be in the course of the radial and cutaneous branches of the musculo-spiral. (See Fig. 32.)

Motor symptoms are widespread and characteristic. There is wrist-drop and inability of all the long extensors of the hand to react if the lesion is high up. There is also inability to extend the elbow. If the nerve is affected in the musculo-spiral groove, the most usual location as stated, the branches to the triceps escape and extension of the elbow is not lost. Lesions below the lower one third of the humerus do not catch the branch to the

supinator longus. The position of the hand (Fig. 30) is characteristic. The fingers can only be extended by the interossei muscles, after the first phalanges are extended, which latter are supplied by the ulnar nerve. The thumb cannot be extended and the fingers can be but a very little better, being progressively diminished from the index to the little finger. Occasionally, on the back of the wrist there develops a synovial tumour due to extreme carpal flexion, and in part due to insufficient support of the extensor tendons, also to involvement of the articular branches of the nerve. This tumour is painless and much exaggerates the wrist deformity. The "grasp" is greatly reduced, due to the extensor loss and unbalancing of the muscle movements. Muscular wasting shows on the dorsal surface of the forearm, and where the supinator longus is involved a peculiar flattening of the forearm is noticed. The triceps may be somewhat wasted.

Prognosis is good in the vast majority of cases, cure resulting.
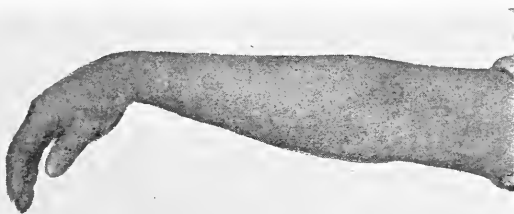
FIG. 30.—WRIST-DROP FROM MUSCULO-SPIRAL ALCOHOLIC PRESSURE PALSY.

Treatment consists in getting rid of the cause, as of alcohol, callus, crutch pressure, etc., or of suturing of the nerve if it has been severed. Ordinarily the use of a blister over the supposed site of lesion is indicated. The arm can be placed in a well-padded anterior splint where there is marked wrist-drop. This will tend to prevent joint deformity in chronic cases. Massage and faradism after the fifth day are indicated in compression cases with separation of the nerve elements. Strychnine in full doses is the drug indicated *par excellence*. In cases of active neuritis the case is to be treated more conservatively as one of local inflammation.

The ulnar nerve, originating from the lowest cervical and first dorsal nerves, and supplying the forearm and ulnar flexor of the wrist, the two inner divisions of the deep flexor of the fingers and all the small muscles of the hand except those innervated by

the median, the abductor of the thumb, and one half of the short
flexor of the thumb, also supplying sensation to the ulnar border
of the hand, including the little finger and half the ring finger,
is when palsied likely to give a wide distribution of symptoms.

Causes.—Neuritis, idiopathic or from trauma, tumours, pres-
sure or separation as by a knife or crockery cut. It may be in-
jured (*a*) above the elbow, (*b*) at the elbow, or (*c*) at the wrist.

Symptoms.—*Sensory* symptoms consist of loss of sensation in
one half of ring and all of little finger, running up the palm and
back of hand to wrist, where it tapers off.

Motor symptoms are marked. The wrist cannot be actively
flexed to the ulnar side, and the thumb is rotated towards the palm
by the abductor, and cannot be adducted. The fingers lose lateral
motion. There is overextension of the metacarpophalangeal joints,
while the unopposed flexors of the second and third are offset by
strong contraction of these digits towards the palm, making the
so-called " claw hand." This flexion is least marked in the index
and middle fingers, which do not lose their lumbrical muscles. The
interosseous spaces are hollowed and the fifth metacarpal bone is
entirely subcutaneous. The hypothenar eminence disappears, but
part of the thenar eminence stands out prominently.

Prognosis is good if the palsy is due to separation as by a knife-
cut and nerve-suturing is done at once. In cases of fracture of the
ulna, with compression, good results will only follow early eradica-
tion of the offending bone or callus. Idiopathic cases of neuritis
are less promising of cure.

Treatment consists first in getting rid of the cause if surgery
will do it, as indicated under prognosis. Acute neuritis should
be combated along ordinary lines. Massage and galvanism are
the most valuable adjuncts to the medical treatment, consisting
of strychnine, the iodides, and general tonic measures. (It is of
interest here to note the sudden restoration of function, especially
of sensation, in case of suturing of the nerve after section, thus
giving a clue to the possibility of nerve force being through some
such agent as electricity.)

Median Nerve.—This nerve arises from the inner and outer
cords of the brachial plexus, and follows the brachial artery to
the bend of the elbow. It supplies all the flexors except the ulnaris,
and the ulnar portion of the deep flexor. It also supplies both
pronators. In the hand it supplies the opponens, abductor, the
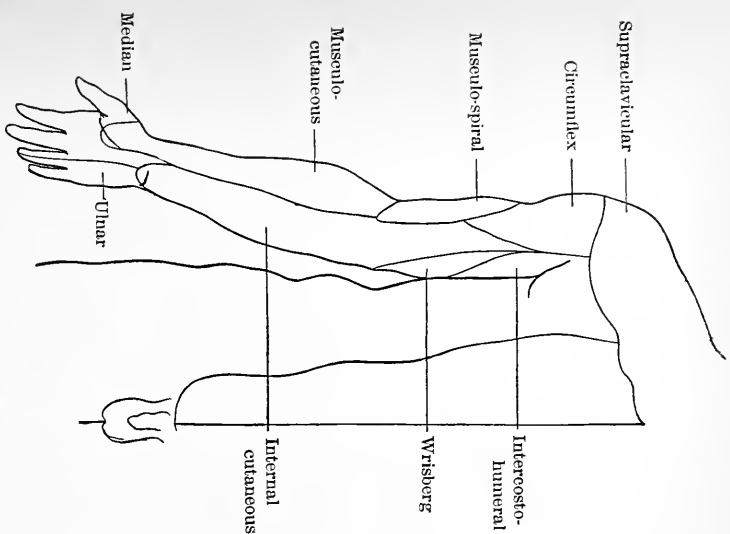
Fig. 31.—Showing the distribution of the sensory nerves of the skin of the arm, anterior aspect.

Supraclavicular

Circumflex

Musculo-spiral

Musculo-cutaneous

Median

Ulnar

Internal cutaneous

Wrisberg

Intercosto-humeral
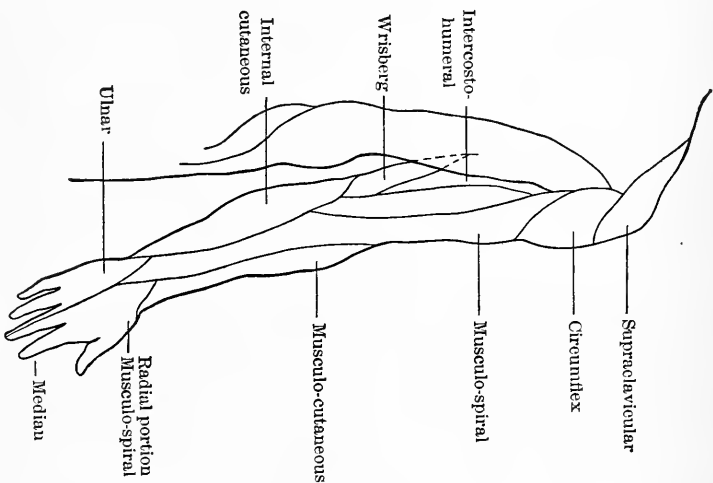
Fig. 32.—Showing the distribution of the sensory nerves of the skin of the arm, posterior aspect.

Internal cutaneous

Wrisberg

Intercosto-humeral

Ulnar

Radial portion Musculo-spiral

Median

Musculo-cutaneous

Musculo-spiral

Circumflex

Supraclavicular
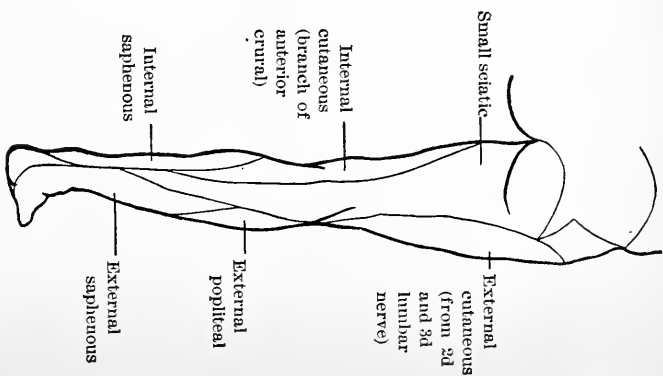
Fig. 33.—Showing the distribution of the sensory nerves of the skin of the leg, posterior aspect.

Small sciatic

Internal cutaneous (branch of anterior crural)

Internal saphenous

External cutaneous (from 2d and 3d lumbar nerve)

External popliteal

External saphenous

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short flexor of the thumb, and the first and second lumbricals. These latter, like the interossei, are accessory to flexors of the fingers (first joints), and are aids to common extensors for extension of the second and third phalanges. The *sensory* distribution of the nerve supplies the radial half of the hand on the palmar side, and the tips of the index and middle fingers on the dorsal aspect.   (See Figs. 31 and 32.)

Cause of Paralysis.—Injury in common with the ulnar and musculo-spiral near the axilla may be the cause of paralysis. Stab wounds near the wrist are not uncommon sources of palsy, also sprains about the wrist, or fractures, as Colles's.

Symptoms, Sensory.—These are usually slight, due to the extensive distribution of the ulnar and radial nerves about the same areas, but would, in complete loss of function, be in the distribution already given.   *Motor:* Pronation is destroyed. The thumb cannot be rolled into the hand or opposed to the fingers. None of the fingers can be flexed except the ring and little fingers, which still act through the ulnar portion of the flexor profundus and interossei. The wrist is flexed slightly to the ulnar side through the flexor ulnaris. The first joints of the fingers are still able to be slightly flexed through the interossei. The hand becomes flattened because the thenar eminence is atrophied and the thumb lies in adduction parallel to the other fingers ("ape hand"). It is the same deformity seen in progressive muscular atrophy of spinal origin.

Prognosis is guardedly favourable if the disease has not gone more than six weeks without proper treatment.

Treatment consists in thorough massage, the use of the ascending galvanic current, 10 milliampères daily five minutes; strychnine, gr. $\frac{1}{30}$ t. i. d., and local protection (splinting) and alteratives, as sodium iodide and arsenic. Nerve suture even late as six months after nerve severance has been attended with good results.

COMBINED PALSIES OF NERVES OF THE ARM

The proximity of the brachial plexus to bony prominences of clavicle, scapula, and humerus makes it liable to injury as a whole; hence, palsy of its various nerve branches may result. The musculo-cutaneous, suprascapular, and circumflex may also be affected secondarily to disease of the fifth and sixth cervical nerves,

from which they arise. Peterson quotes from the Revue Neurologie, December 15, 1900, that forcible dragging of the arm up or down may lacerate the anterior nerve-roots.

Or a neoplasm or injury opposite the sixth cervical vertebra may affect both nerves. At this point (between the scaleni muscles) Erb has found that the circumflex and suprascapular nerves can be stimulated by electricity. The muscles involved in such a palsy are the spinati, deltoid biceps, and brachialis anticus, while the sensory disturbance corresponds to the cutaneous distribution of the nerves affected.

Injuries to the *brachial plexus* are usually severe. I have never seen a crush involving the branches recover. As a rule, injuries involve all its branches. But single nerves—e. g., the ulnar—may be affected; or the ulnar in association with the nerve of Wrisberg, etc., in which case anæsthesia along the entire inner aspect of the arm will also accompany the motor palsy. The so-called "birth palsies" are due to traction on the arm, or to the use of a hook in the axilla or to head forceps during delivery; and usually involve all the branches—viz., the median, ulnar, and musculo-spiral. A fracture of the humerus may injure the musculo-spiral and ulnar nerves. The radial and ulnar nerves may be simultaneously injured in fractures in the forearm or disease at the wrist. Infected wounds of the hand may produce ascending neuritis, which may ultimately affect the entire brachial plexus (*neuritis migrans*).

Pott's or other disease of the vertebræ may affect the brachial nerves secondarily, as also may meningitis, particularly the hypertrophic form; or even disease of the cord, as syringomyelia, may do the same thing.

Prognosis has been indicated.

Treatment will consist of getting rid of the cause at once if degeneration and an ill result shall not follow. The other measures to be adopted will be described below.

Brachial Neuritis.—Gowers compares this with sciatic neuritis. The symptoms consist of pain and tenderness in the distribution of the branches, particularly when the shoulder is moved, since the circumflex is then much disturbed as well as the other nerves, though to a less degree. The pain is often mistaken for neuralgia, for rheumatism of the shoulder or arm; careful examination will eliminate these. Women suffer more frequently than men. The

disease occurs oftener after middle life, especially in gouty or
rheumatic people. Lumbago or sciatica may also be existent in
the case. I have seen a needle prick in the finger in two cases
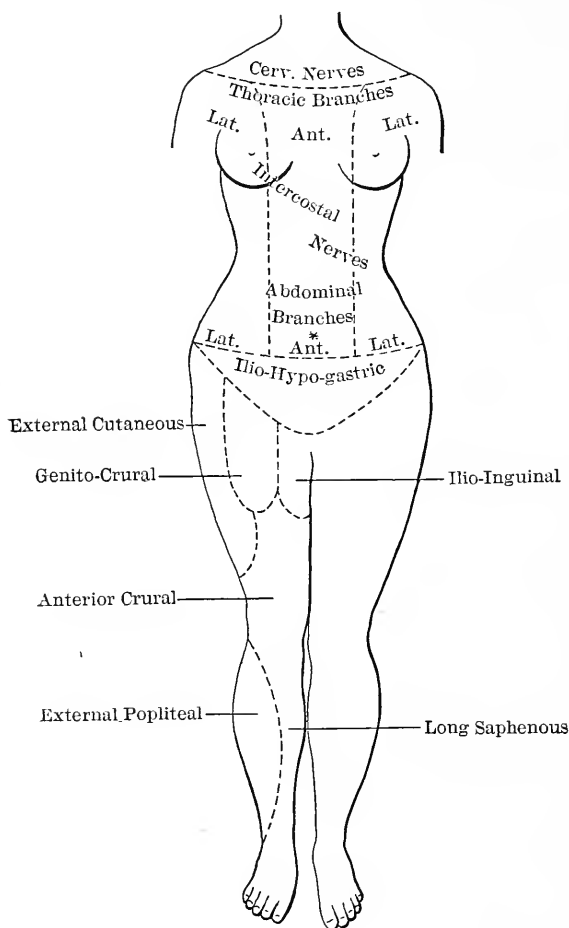
FIG. 34.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE SKIN,
ANTERIOR ASPECT OF TRUNK AND LEG.

produce most profound brachial neuritis. The entire arm becomes
wasted and the hand flattened. The muscles as well as the nerves
are tender, and the slightest movement gives severe shooting pain
in the arm and neck. Reaction of degeneration is found. The

reflexes are usually diminished, but as in chronic sciatica may rarely be exaggerated.

The *prognosis* is most serious as to cure, relapses being common in the most favourable cases.

Treatment consists in rest of mind and body as fundamental principles.   The lithæmic state must be looked to.   Counter-irritation along the course of the plexus and nerves by the actual cautery affords great relief.   Dry heat will often do good.   Cold usually aggravates.   Protection of the arm by a splint or cotton wool is a valuable remedial measure.   Ascending galvanism is of value.   Amputation of the arm below the insertion of the deltoid is the only treatment for severe laceration of the brachial plexus.   In this way the incumbrance of a totally palsied arm is done away with, to the comfort of the patient.

NERVES OF THE TRUNK

These nerves supplied by the thoracic and two upper pairs of lumbar nerves are rarely singly involved.   Pott's disease may affect them, but the loss is slight unless several nerves are affected simultaneously.   The girdle sense of tabes or neuritic pain of vertebral disease are diagnosticated by observing other signs of the respective diseases.   Herpes zoster may encircle the body, and in such cases the symptomatic disorder is significant of serious root mischief either due to extension from spondylitis or to sclerotic involvement, as in tabes; or it may be from direct metastasis from a neoplasm.   Unilateral herpes is rather characteristic of gastro-intestinal disorder only.   Wide-spread trunkal herpes is, as a rule, due to final disease of the gray matter rather than of the nerves themselves.   (See Figs. 34 and 35 for cutaneous nerves of trunk.)

NERVES OF THE LOWER EXTREMITY

These are not so frequently involved as those of the arm, but are subject to special disease through pressure in pelvic growths, and in females by pressure upon the sacral plexus during labour.   Inflammation of pelvic viscera or psoas abscess may affect the plexus. (See Figs. 33 and 34 for cutaneous nerves of lower extremities.)

The external cutaneous branch of the anterior crural nerve supplying the outer side of the thigh is very liable to disease.   A queer condition termed *paræsthetic meralgia,* consisting of a sense

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of burning (causalgia) in the outer upper aspect of the thigh, is found in neurasthenic women, especially when secondary to ovarian disease; probably caused by a reflex reference through irritation of this sensory branch.   In other cases a neuritis has been set up.
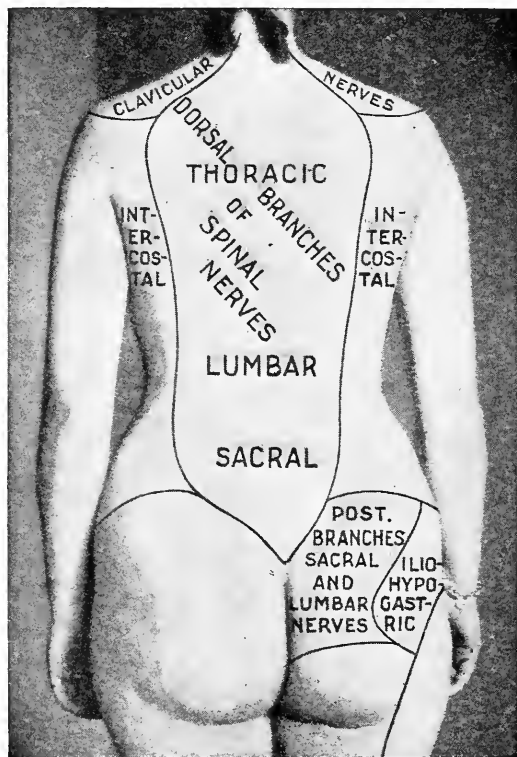

FIG. 35.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE SKIN. POSTERIOR ASPECT OF TRUNK (Butler).

Gout, rheumatism, excesses in tea-drinking, in alcohol or tobacco may cause it, or as a resultant of multiple neuritis this peculiar hyperæsthesia may continue for months or years.

Treatment consists in treating the neurasthenia and in nerve-stretching, which usually is successful.

Anterior Crural.—This nerve may be affected (*a*) within the abdomen, when flexion of the thigh on the abdomen is weakened because the iliacus muscle is included, and (*b*) below Poupart's ligament.

Symptoms due to cutting off of entire function, are wasting of the quadriceps and abolition of the knee-jerk. *Anæsthesia* will be found to extend from the groin to the inner side of the foot. There is but a narrow strip of normal sensation remaining, which runs down the back of the thigh below the knee, spreads out over the external portion of the calf, embracing the foot except on the inner aspect. (See Figs. 33, 34, and 36.)

Prognosis is good if the cause can be eradicated. When due to pelvic tumour, it is hopeless; but disease of the internal genitalia, if causative, being eradicated will relieve the secondary neuritis. Idiopathic cases must be treated as neuritis elsewhere. Applying a long external splint is of great service. Galvanism and massage are valuable after acute symptoms have abated.

Obturator Nerve.—This nerve comes from the same source as the anterior crural nerve. It supplies the adductors of the thigh, also the knee and hip-joints. Hence it is that " referred " pain may be to the knee in cases of coxalgia or disease of the knee may be referred to the hip of same side.

Causes.—Prolonged horseback-riding may cause the neuritis, or tearing of the muscles by athletes; or a direct trauma may produce neuritis of the obturator.

Symptoms of paralysis of this nerve. Proper adduction of the thighs is prevented; there is impaired external rotation of the thigh through palsy of the external obturator and pectineus muscles. Wasting occurs on the inner aspect of the thigh above. *Sensory* symptoms consist of knee or hip pain, depending upon whether one or both branches are involved.

Treatment consists of rest, massage, galvanism, and relief of pain by anodynes or by hot applications of lead water and laudanum, twenty-five per cent. Disease of knee or hip should be treated.

The superior gluteal nerve may be inflamed, and this causes palsy of the gluteus minimus and medius and the tensor vagina femoris muscles. The *symptoms* consist of weakness of abduction, outward rotation, and circumduction of the thigh.

The Great Sciatic Nerve.—The size and exposure of this nerve and its branches make it more liable to disease than any other single nerve of the lower extremity. It supplies the knee flexors, which are also partial extensors of the thigh upon the body. It also applies all muscles below the knee. (See Figs. 33, 34, and 35.)

Symptoms.—From the peculiar mechanical position of the mus-
cles palsied in disease of this nerve, the disability to the patient
is less than that resulting from disease of the anterior crural
nerve above recited. The paresis of muscles supplying only *about*
the large joints of the hip and knee is the explanation of this.
The leg is carried forward in progression as a " pegleg " similar
to that of the hemiplegic gait. Wasting is marked. Perforating
ulcer of foot may follow. *Sensory* phenomena consist of anæs-
thesia occupying the narrow strip of the thigh posteriorly and the
outer aspect of leg and foot described in the last section.

Disease of the **external popliteal or peroneal** nerve may be lo-
cated (*a*) in the ham or (*b*) below the knee on the outside of
neck of the femur. This branch nerve supplies the long extensors
of the toes, also the peronei muscles.

Symptoms, Motor.—Foot-drop with toe-drop follow; and
equinovarus may result from contraction of the unopposed flex-
ors. There is much wasting about the fibula and in the anterior
group of leg muscles. (See also Sciatic Neuritis.)

The internal popliteal nerve supplies the calf muscles, the long
flexors of the toes, all the small muscles of the foot save the short
extensors of the toes. If its branch, the *internal* plantar, is di-
vided, there results a paralysis of the short flexor muscles of the
toes, the plantar muscles of the great toe, except the adductor,
and the two inner lumbricales. " Hammer-toe " results from ex-
tension of first joint and flexion of second joint. *Sensory* symp-
toms consist of anæsthesia in the outer plantar surface of half the
sole, one half of the fourth, and all of the fifth toe. (Fig. 36.)

If the *external* plantar branch is alone involved, the symptoms
are loss of power of the muscles of the little toes, the two outer
lumbricales, all the interossei, and the adductor of the great toe.
The hammer-toe deformity is found in all the toes. *Sensory* symp-
toms then consist of anæsthesia in the outer plantar surface of
half the sole, one half of the fourth, and all of the fifth toe.

If the *internal popliteal* is divided in the popliteal space in
addition to above symptoms, there is inability to rotate the flexed
leg, due to palsy of the popliteus muscle; and also a loss of power
of extending the foot. Talipes calcaneus may therefore result
with, in addition, the arch of the foot becoming exaggerated.

Morton's Disease, or Metatarsalgia.—I have seen a number of
cases under the discoverer's care. Dr. Morton considered it due
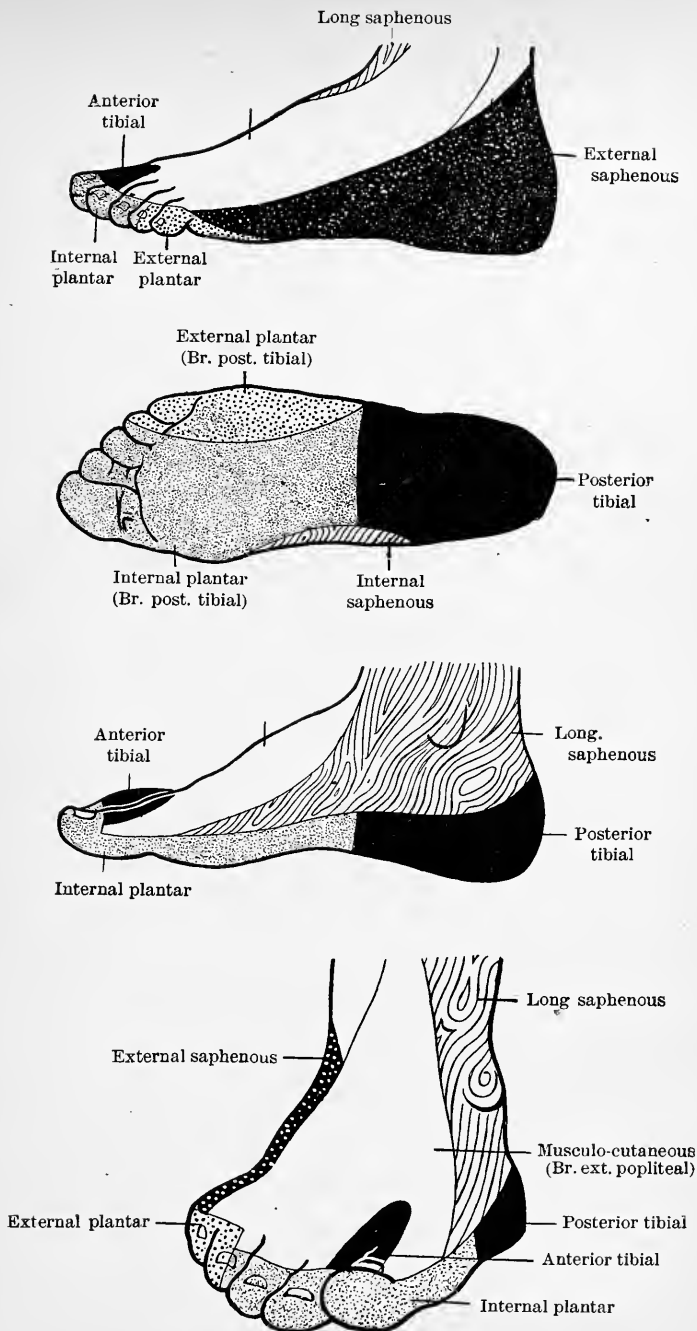
FIG. 36.—Showing the Distribution of the Sensory Nerves of the Skin of the Foot (Butler).

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to pressure neuritis of a filament of a digital branch of the external plantar nerve lying between the heads of the metatarsal bones. Narrow shoes may favour its development, as may gout.

The *symptoms* consist of sudden excruciating pain in or near dorsum of foot opposite the fourth toe. This pain shoots up the leg, and may be relieved by firm pressure, as of a lace shoe; or by placing in the boot a " sole plate " as manufactured by Gefvert.

Prognosis in gouty cases is, however, bad.   Excision of the head of the fourth metacarpal bone is the radical measure that we have seen effect a cure in several cases.

Treatment of palsies of the various branches of the sciatic nerve outlined above consists in nerve suture for division; of getting rid of adjacent local disease, as tumours, fracture, or callus; and in the treating of any diathetic tendencies.   Other treatment useful is described under Sciatic Neuritis.

RHIZOMELIQUE SPONDYLITIS

This disease consists of an osteo-arthritis of the spine of unknown origin; hence it can be set down as probably dependent upon trophic disturbance through the nervous system.   Deformity may result, depending upon whether the interarticular vertebral fibro-cartilages are absorbed or not before the proliferation and ossification occurs about the respective joints.   When the disks are absorbed quickly, kyphosis, scoliosis, and other deformities of the spine follow.   The large joints near the body are next attacked, such as the hip, knees, and shoulders.   The Roentgen rays show bony exudate and the absorption of cartilages as described.

Ætiology is not definite.   Heredity, cold, wet, and trauma or gonorrhœal infection are all supposed to be factors in individual cases.   But the above causes may be but exciting elements, the disturbed metabolism from lowered nutrition being the predisposing cause.

Symptoms.—These are entirely dependent as to the nervous system upon trophic disturbance noted (also atrophy of muscles may occur as in two cases that are under the writer's care at the Philadelphia Hospital at the present time) or upon whether spinal nerves or spinal cord are encroached upon by pressure, etc.   Pain and limitation of motion are usually prominent symptoms.   The pain may be severe and be " referred," or occur at the site of the spine disease, usually then at anterior aspect in the back of abdo-
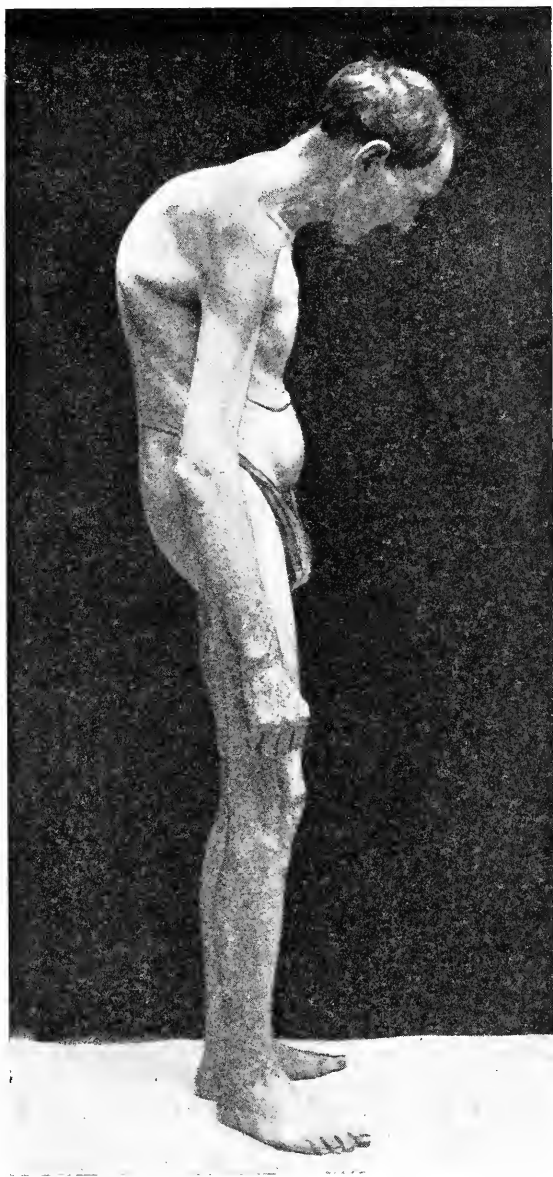
FIG. 37.—RHIZOMELIQUE SPONDYLITIS (DORSAL SPINE).
(Medico-Chirurgical Hospital.)

men.   It is worse after sleeping, probably due to sluggish circula-
tion during sleep producing stagnation of circulation about a dis-
eased area; also to metabolites irritating the sensory nerve roots.
" Girdle pains " are one-sided usually, because one side of the
vertebral column is first attacked.   There is impairment of mo-
tion in the legs; or, indeed, complete paraplegia may result.   Close
study may reveal areas of hyperæsthesia, hypæsthesia, or anæs-
thesia depending on the lesion being at the time an irritative or
destructive one.   Coughing or movement of the head or trunk will
bring on severe pain.   According as the erosion of cartilage and
osseous deposit predominates or muscular spasm is productive
of the rigidity or deformity, will they be of more or less permanent
nature.

   Prognosis.—Ultimately the patient may become entirely bed-
ridden and helpless save for the use of the smaller joints.   Occa-
sionally the disease is stayed from advance by timely hygiene and
treatment.

   Treatment consists of constitutional and hygienic measures.
Sirup hypophos. comp., ʒi t. i. d., or cod-liver oil in cold weather
are valuable nutritional drugs.   Sod. salicylat., gr. x t. i. d., or
potassium iodide, gr. x t. i. d., or syr. ferri, iodidi, a half drachm
after meals, are respectively the best antirheumatics, alternatives,
and hæmatics that can be employed.   Look to the kidneys and the
eliminative functions generally.   A toxic process in the intestine
may aggravate, hence the use of salol, gr. ii, in elix. lactopeptine,
a drachm, thrice daily, may be desirable.   The digestive tract
must also be conserved in these patients and full simple diet given,
including abundance of eggs and milk.   The orthopædic surgeon
may aid by bloodless operation of extension or through applying
apparatus, or by use of a wheel crutch to get the patient about
on his feet.   I have had one case thus bettered, so that from being
bedridden he could walk about.   His general health was much
improved.

DISEASES OF THE MEMBRANES OF THE BRAIN

WE shall consider here anæmia, hyperæmia, inflammation of the dura mater (pachymeningitis), and inflammation of the pia mater (leptomeningitis).

Anæmia of the membranes of the brain cannot be well separated from anæmia of the encephalon itself, and must therefore be studied with anæmia of the brain.

Hyperæmia must likewise be considered with hyperæmia of the brain so far as that of the pia is considered. Dural congestion or hyperæmia may occur as the result of injuries, of sunstroke, of poisons such as uric acid, or of infectious diseases, such as measles, scarlet fever, and syphilis; the symptoms being like those of pachymeningitis of the congestive stage, which will be described under that heading. The principal symptoms, are, however, vertiginous attacks and peculiar sensations of fulness in the head. The treatment is the same as for the onset of meningitis.

INFLAMMATION OF THE DURA OR PACHYMEN-INGITIS EXTERNA

Since the so-called pachymeningitis interna is probably a hæmorrhagic disease (or hæmatoma of the dura mater), it will be described under the head of dural hæmorrhage. It is extremely rare to have a true simple internal meningitis of the dura. (See p. 175.)

Pachymeningitis externa first involves the outer surface of the dura. There are less pressure symptoms than in hæmatoma.

MENINGITIS

This implies inflammation of the membranes of the cord and brain, being divided into pachymeningitis (dura) and leptomeningitis (pia), each in turn being divided into *acute* and *chronic*

simple meningitis; and secondarily into *acute* and *chronic* infectious meningitis, the latter including infectious cerebro-spinal meningitis, tubercular, leptothrix infection, and the various streptococci infectious forms. Various other organisms produce inflammation of the membranes, the principal ones, however, being included in the above list. Usually when we speak of meningitis we refer to meningitis of the dura, so that specifications should be made of these inflammations for the sake of exactness.

ACUTE CEREBRAL PACHYMENINGITIS

Inflammations of the Membranes of the Brain.—This may be *primary* or *secondary*, the *former* being caused by the so-called " catching cold," or exposure to cold, or exposure to extreme heat. The *causes* of the *latter* form are infectious diseases, such as pneumonia, typhoid fever, infectious cerebro-spinal meningitis, injuries, carious bone as from mastoid disease, syphilis, erysipelas, etc.

Symptoms.—This disease is usually ushered in by a marked chill, followed by, as a rule, hyperpyrexia, the temperature running up to 104° or 106° F. The patient may suddenly pass into convulsions of tonic and clonic nature, with retraction of the head, delirium, mania, and hyperemesis, frequently of the *projectile* type. The convulsions may be localized or at least begin in one member, due to irritation of a particular centre over the motor cortex. Paralysis may follow. The pupils usually are irregularly dilated. There may be amblyopia, due to optic neuritis, which is soon set up in severe cases and accompanied by marked swelling of the disks. Various other palsies may occur, producing internal strabismus or external strabismus, frequently accompanied by diplopia. It is very rare that hemianopsia follows. Hearing may be affected in the beginning, it being *acute* as is the case with the other special senses. Late in the disease deafness or dulness of hearing may occur. Localized facial palsies may also follow spasm of these muscles, due to the involvement of the nuclei of the facial nerves. If the basilar membranes are involved bulbar symptoms may supervene; or a deafness, or blindness in the most serious cases. The *duration* of acute pachymeningitis will depend upon the degree of infection or the resisting power of the patient. Death may follow early severe convulsions. The acute

symptoms may last a week or ten days, when termination by crisis may occur.[1] Or, as is the rule, a gradual defervescence of the fever and recovery with more or less sequelæ, depending upon the severity of the disease.

Prognosis.—In children it is worse than in adults, owing to the lack of resistance through immature development of the nervous system in them. Occasionally a case will recover with apparently absolute integrity of the nervous system and no sequelæ whatever, but in the vast majority of cases some defect is left behind, either blindness, due to progressive atrophy of the optic nerves, or deafness from involvement of the acoustic nerves, etc. These two special senses are the most frequently involved, although the others, but very rarely, may also be affected. Sequelæ, as hemiplegia, paraplegia, or monoplegia of spastic type, depending upon the degree of involvement of the motor cortex, are frequent.

Treatment.—This will depend entirely upon the nature of the cause. If primary disease or any septic foci exist in the body, these should be dealt with at once. (The treatment of infectious cerebrospinal meningitis will be considered in its proper place.)

The patient should be, as a rule, placed in a dark room, in quiet surroundings, and well protected from atmospheric exposure. Such measures are necessitated by the extreme hyperexcitability in the beginning of the disease. Hyperpyrexia should be treated by antipyretics guardedly administered, by the sponge-bath or ice-pack, and in some cases of plethoric individuals venesection should be tried. The application of leeches to the temple or wet cups to the nape of the neck are valuable methods of depleting the congested meninges; to-day not enough used. For the control of convulsions, the antipyretic measures detailed may be sufficient in the milder cases, but in the more serious ones the use of large doses of bromides and chloral may be necessary. If these cannot be retained by the stomach they should be given per rectum, and in twice the dosage as given by the mouth. If heart failure occurs later in the course of the disease, cardiac stimulants may be necessary, such as digitalis, or in acute syncope hypodermics of ether or strychnine may be necessary, continued until the crisis is past. In case of injury or abscess, surgical help must be sought.

[1] Kernig's sign may be present in meningitis—viz., the inability to extend the legs while sitting, but ability to do so when recumbent. It is *not* pathognomonic of the disease.

Potassium or sodium iodide are valuable remedies for the absorption of the exudate. If betterment of ocular troubles does not occur with this, the destruction of the cranial nerves passing out at the base of the brain may be inferred and a basilar type confirmed. The treatment of sequelæ, as optic atrophy, etc., will be the province of specialists in this department of medicine, to whom the case should be early referred. Epilepsy may be a sequel, and should be treated symptomatically if the original source of irritation cannot be removed.

Causes of Chronic Meningitis.—This is generally the result of the acute form. Some cases are so insidious that they are designated subacute or chronic from the first discovery of symptoms. Sunstroke is quite frequently a cause of this type, as may be lues or trauma.

Symptoms.—Persistent headache of a dull character, worse when the patient is exposed to heat or when he stoops forward or lies down. He also complains of pain in the neck, frequently radiating towards the shoulders, and may suffer from general paresis of the extremities, usually of a spastic nature. Or he may be the subject of deafness, vertigo, or optic atrophy. Such patients, too, may be subjects of secondary epilepsy.

Diagnosis.—This is to be made from chronic cerebritis, tumour of the brain, hydrocephalus. In cerebritis there is usually more mental failure or even dementia with less spasticity of any existent palsy. In tumour of the brain the special predominance of optic neuritis (95 per cent), together with localizing symptoms, will differentiate, while in hydrocephalus, the enlargement of the head of the peculiar metallic sound on percussion, or the exophthalmos, are points for differential diagnosis.

Prognosis.—This is usually bad as to permanent recovery, the patient being liable to exacerbations at any time, due to catching cold or exposure to heat. In adults the chronic form of meningitis may exist for many years. In *children*, since interference of development of the brain follows, life is materially curtailed even in the milder cases, such patients usually succumbing early to intercurrent disease. Blindness and deafness occur in the chronic form as the result of the acute type, and as a rule such cases are practically incurable. Imbecility is not unusual.

Treatment.—This will simply consist of additional measures to those already referred to—namely, the treatment of the convul-

sions or of contractures and of palsies, which latter may require the services of the orthopædic surgeon. Massage and Swedish movements should be given a fair trial in these cases. If a large exudate, especially of the hæmorrhagic type, or a localized blood tumour of the dura exists, the surgeon may by prompt operation save the patient's life, as I have known in one case. Extensive counterirritation over the scalp is of value.

CEREBRAL PACHYMENINGITIS INTERNA HÆMORRHAGICA OR HÆMATOMA DURA

This is a type of local inflammation involving the internal layer of the dura. It is more frequent in male adults and in those subject to chronic alcoholism or prolonged intoxication.

Symptoms are insidious, the patient complaining of pain in the head, following an alcoholic debauch. There are manifest symptoms of dementia, such as loss of memory, incoherent speech, with periods of excitement. Frequently the patient will pass into an uræmic state, which is due no doubt to kidney insufficiency at the time, even though the kidneys may not be permanently disabled. The case may recover from the uræmic attack rather suddenly. Usually within a few days or a week recurrence of symptoms occurs, such as added weakness of the extremities, more pronounced upon one side of the body. Fifty per cent of the hæmorrhages are on one side of the dura. The mental obtundity deepens, the patient passes into delirium. There is also noticed general tremulousness. It will be found, too, that the paralysis is much more *spastic* than the rapidity of onset and the extent of motor weakness would warrant. This is a point of value in making the diagnosis of this serious malady. A distinct point in the symptomatology is, therefore, exacerbations and remissions without profound coma at any time until late in the disease.

Diagnosis.—This disease is so difficult to diagnose that it seems useless to set down differential points. Ingravescent hæmorrhage into the cerebrum may be mistaken for it, as well as uræmia, indicated above. From the former it may be distinguished by the more exact symptomatology of true apoplexy in the latter. From uræmia, the urinary examination would be of great value, since the lesser bulk of albumin, if any, would be found in a meningitis.

Prognosis.—This is absolutely bad. The patient is usually car-

ried off within ten days after the onset of symptoms. Death generally occurs not alone perhaps from the hæmorrhage, which is usually *massive* and spread over the entire cortex of one side, but very likely it is completed by the general alcoholic toxæmia existent in these patients.

Treatment.—This consists, therefore, in the same form as the acute meningitis given above. *Surgical treatment* is not as yet
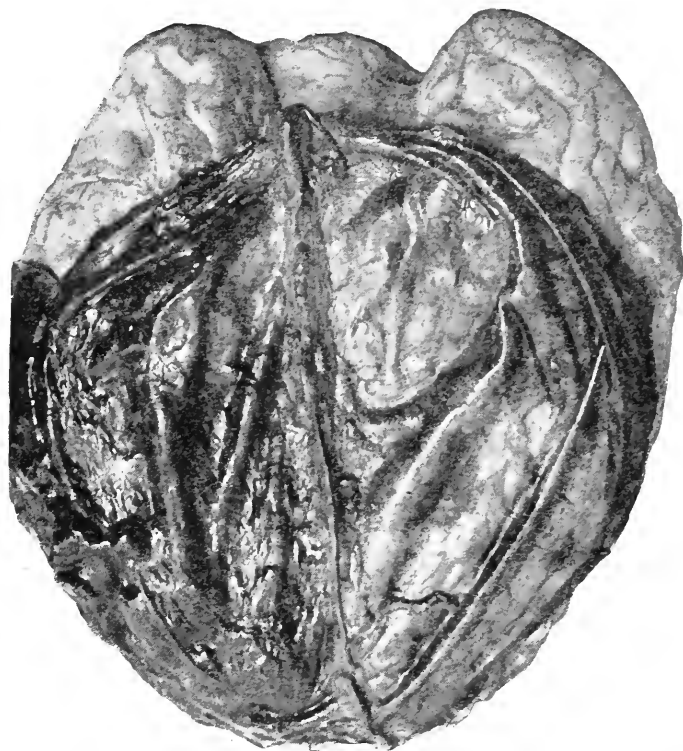

FIG. 38.—HÆMORRHAGIC PACHYMENINGITIS INTERNA (ALCOHOLIC). Specimen from a case in the Philadelphia Hospital. (Dura is reversed in the photograph.)

fully developed, and has not been resorted to in many cases, but would seem to be the only possible chance of saving the patient's life. If it were not for the extreme asthenia, a diagnosis could be made and the surgeon's skill in trephining and removing the clot might save some cases. Venesection and transfusion of normal salt solution is a treatment that should be resorted to in

cases of marked toxæmia. Supportive measures should, of course, be insisted upon in all these cases.

CEREBRAL LEPTOMENINGITIS

This is *usually* an *acute* infection. It is, as a rule, secondary to some septic foci, as abscess of the lung, liver, etc.

Symptoms.—These are fulminating in character, accompanied by high temperature, great mental anxiety, and a hectic type of temperature curve, which remains, at about 104° or 106° F. Convulsions are not so common, nor spactic rigidity of muscles so frequent as in the acute form of pachymeningitis, nor is intense head pain of so great importance as in the former disease. Localized palsies of sudden development are rather frequent, depending upon exudation over a motor centre. Duration of the disease is from a few days to ten days, the patient usually succumbing with the hyperpyrexia or from the general infection. Termination by crisis may occur, or a chronic septic inflammation of the brain may result.

Prognosis.—This is bad in the majority of cases. Death of the patient usually takes place within seventy-two hours. In many cases a remote *abscess* in the brain may be the result of the above disease.

Treatment.—This would not be different from that for pachymeningitis excepting that it is more incumbent to seek for septic foci both in the brain and in other organs, to which it is so frequently secondary.

HYDROCEPHALUS

This is a condition, congenital or acquired, in which there is an accumulation of the fluid within the ventricles of the brain. There are two *types* of hydrocephalus, *external* and *internal*. It is again divided into *acute* and *chronic*, although the chronic form is the more common variety. The internal type implies an excessive collection of fluid within the ventricles, whereas the external type refers to cases in which the fluid is mainly in the subdural space. Usually the two are coexistent, but internal hydrocephalus always predominates.

Symptoms of acute hydrocephalus may follow acute inflammation of the ependyma. It is more usual in babyhood and in

children suffering with marasmus, in which subjects there seems to be a special tendency to local inflammation of this serous lining of the brain. Symptoms are those of subacute meningitis, excepting that convulsions are not so frequent and the temperature is never so high as in the former disease. The child is usually in a condition of muscular rigidity, however, the attack itself generally being induced by autointoxication. In addition to the muscular rigidity with tetanic spasms, at times there is evident an enlargement of the head, bulging of the fontanelles, and depression of the eyeballs within the socket, together with exophthalmos. The vault of the cranium projects out in proportion to the rest of the skull, so that there seems to be enormous enlargement above the ears. In some cases the sutures are seen to be separated and the Wormian bones palpable. The " hydrocephalic cry," consisting of a long, deep respiratory sound, is a very usual symptom. Percussion over the skull will frequently produce a pseudo-tympanitic note, and on auscultation over the skull a distinct bruit can be detected synchronous with the pulse-beat. The patient may finally pass into a condition of tetany and die from exhaustion.

Pathology consists of subacute ependymitis with excess of serum within the ventricles, which may, as indicated, also extend to the arachnoid.

Prognosis.—This is bad, as a rule. In the acutest forms life becomes extinct within a few months. Occasionally the symptoms abate, and the patient is left with the chronic enlargement of the head which, if not too extensive, will not produce paralysis of the extremities nor any considerable degree of mental impairment, the rule, however, being that cases will result in imbecility with the general symptoms of spastic paralysis of childhood. It should be remembered that this is frequently a congenital condition, which is lit up soon after birth. It is astounding to what degree the convolutions may be pressed upon, and life and fair mentality be preserved in a small number of cases.

Treatment is hygienic, medicinal, and operative. Prophylactic measures should be part of the treatment—nourishment of the child predisposed or born with the condition is most essential. Inunctions of cod-liver oil, with the idea of gaining weight and greater resistance to diseases, is of value. Spasms should be controlled by chloral and bromides. Sodium iodide is especially of service in infants, being less liable to disturb the stomach. It

should be given in two-drop or three-drop doses of a saturated solution, gradually increased to the physiological limit. Some advise compression of the skull by proper bandaging with the idea that this may tend favourably against the formation of exudation. Should symptoms of compression develop, however, the bandage should be removed immediately. Surgical aid through craniectomy, or tapping the ventricles, under antiseptic precautions, may be of value as a *dernier ressort.*

Chronic Hydrocephalus.—This is usually the result of the acute form of hydrocephalus, and from the evidence found at birth in same cases, it is apparently chronic from the very first—i. e., it has existed some time in the prenatal life of the child. The signs here may first be noted during labour, an enlarged head being the cause of dystocia. More frequently, however, the hydrocephalus becomes apparent some time after birth.

Causes of this condition are not definitely known. The pathology offers no direct clue to the nature of the trouble. The lateral ventricles are greatly distended, but the ependyma is usually clear, occasionally granular and thickened. The chorioid plexuses are extremely vascular. The third ventricle is dilated, as is also the aqueduct of Sylvius and the fourth ventricle. The quantity of fluid may be several litres. It contains salts, albumin, etc.

The cerebral cortex is greatly stretched, and over the middle region it may measure but a couple of millimetres. The basal ganglia are flattened. The bones are extremely thin, and the Wormian bones themselves may gradually extend, due to the effect on the part of nature to fill in the gap of the widely separated edges of the bones.

Symptoms are similar to those of the acute type, although not so severe. Convulsions may occur, reflexes are increased, the child may learn to walk late, but ultimately usually becomes feeble and very spastic. The mental condition is widely variable, from that of the ordinary imbecile to that of average intellect.

Diagnosis.—The rhachitic head should be told from that of acute hydrocephalus, and is known, too, by the other evidences of the enlargement of the epiphyses of the long bones. The head in rickets is usually square, and not round or globular. The separation of the bones of the skull is not usual, nor is there the tympanitic note produced on percussion over the skull nor the bruit heard on auscultation, as in hydrocephalus.

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The *acquired form of chronic hydrocephalus* is simply the result of acute hydrocephalus, but is said by some writers to be primary, essential, or idiopathic; that is to say, it comes on spon-
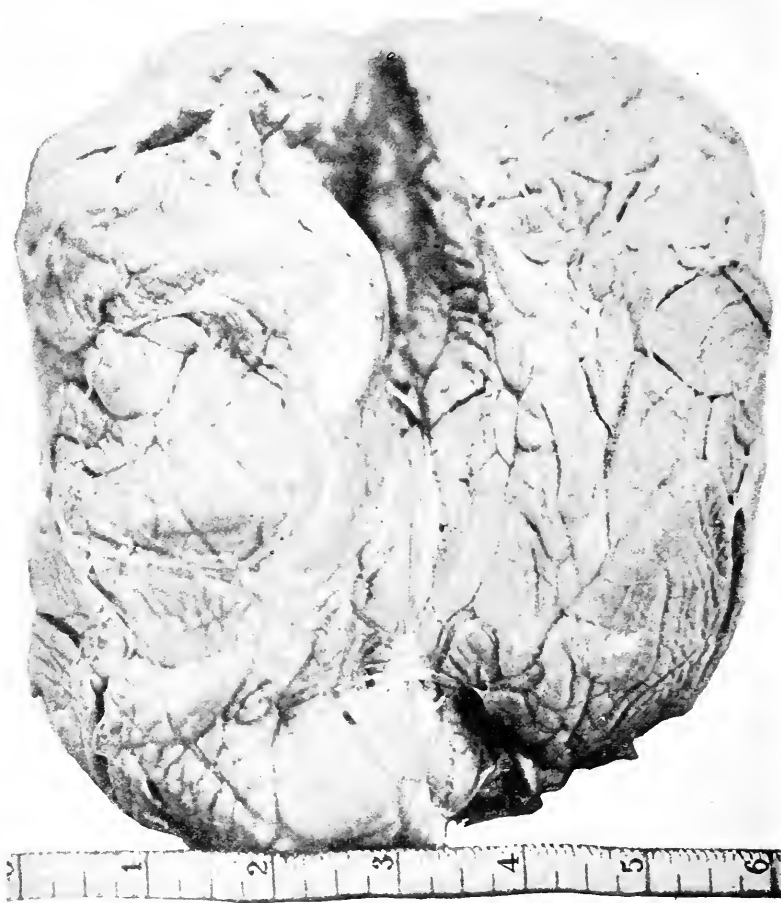
FIG. 39.—BRAIN. CASE OF HYDROCEPHALUS IN A CHILD OF FIVE YEARS. Size indicated in inches (reduced).—(Howard Hospital.)

taneously in the adult without observable lesion. Osler quotes the statement of Mr. Whiteway that Dean Swift was a subject of this variety, but doubts the possibility, and assumed that his case was

probably one of a spurious form of hydrocephalus, *ex vacuo*; that is, within the arachnoid alone. The *symptoms of the acquired* form would vary with the time in life of its development. If developing early, the symptoms already rehearsed will be present; if late in life, the patient will suffer from headaches, the gait *becoming gradually* irregular and ataxic; with a spasticity, how-
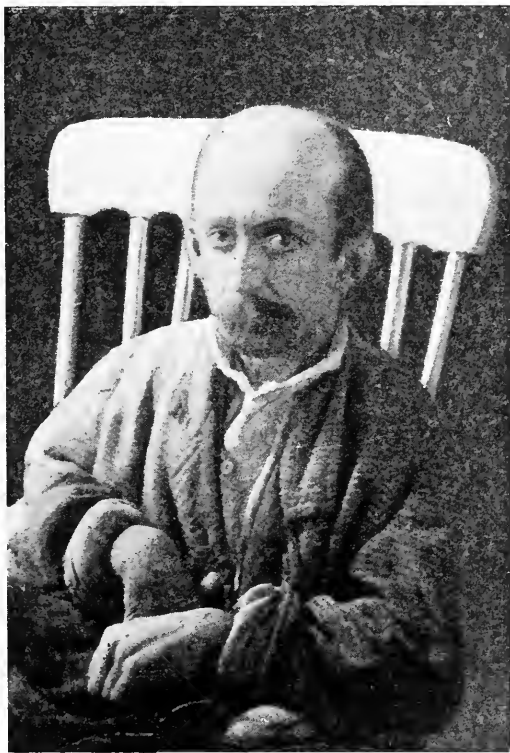
FIG. 40.—SPASTIC DIPLEGIA FROM CHRONIC HYDROCEPHALUS (ADULT MALE).
(Philadelphia Hospital.)

ever, which distinguishes it from other forms of gait. In some cases there will be prolonged attacks of coma with slow pulse, due to irritation of the *vagi* nuclei by the pressure. One case on record is reported to have been unconscious for three months. Optic neuritis soon supervenes, and in the worst cases rapidly progresses after once begun. Chronic purulent ependymitis may precede death in some cases.

Meningitis serosa is a type distinguished by Quincke, and consists of serous meningitis as distinguished from ordinary *leptomeningitis* and from hydrocephalus. Quincke describes the affection as coming on suddenly in children with intense pain in head, signs of intracranial pressure, slow pulse, and choked disks. Retraction of the head exists, without fever, however. Some cases are much more insidious and are frequently mistaken for brain tumour.

Treatment consists in the relief of pain and in making an effort to cause absorption of the collected fluid. This will be best done by giving depletive remedies, such as gamboge in *sthenic* individuals, or podophyllin or even calomel in repeated dosage. For the more usual *asthenic* cases such harsh treatment cannot be carried out, and we must depend upon free diuresis, producing increased action of the skin through such drugs as pilocarpine. Potassium iodide is a valuable remedy as a sorbefacient. Bichloride of mercury, added to the above iodide, may favour resorption of the exudate within the brain. As a prophylactic measure the patient should be carefully guarded against inclement weather and given nutritious diet; and mental strain should be avoided. Skilled massage may be of value in these cases.

CHAPTER X

DIFFUSE AND LOCAL DISEASE OF THE BRAIN

Cerebral Localization (Epitomized).—It must be remembered that cerebral localization is not an exact science, because symptoms may also be produced by compression, causing irritation, paresis, or paralysis of adjacent centres; also, that circulatory disturbances occurring in the course of disease of the membranes of, or of the encephalon itself, may cause wide-spread functional disturbance that disobey all scientific rules.[1] Still, in the majority of instances, close day-to-day study of the case will be rewarded by brilliant diagnoses, and help, therefore, for the patient. The accompanying illustrations (Figs. 41 and 42) give the respective locations of the definitely know *centres* in the cerebrum. Beginning from the posterior aspect, we will enumerate the function of each centre and lobe (for fuller description, see Chapter I, on Anatomy, etc.).

Occipital Lobe.—Here lie the centres of vision, destruction of which causes cortical blindness (in the absence of lesion of the eye or optic nerve). Hemianopsia may also result as indicated under diseases of the *optic nerves*. Sensory centres are also present in the occipital lobe. (Cortical centres concerned in speech are described under Aphasia.)

Temporal Lobe.—Here lies the centre of audition—i. e., for word hearing—in the posterior part of the first and second convolutions—and the centre for *naming* in the third convolution.

The *central convolutions* of the parietal and frontal lobes of the cerebrum are principally centres for voluntary motor function. The upper portion of the first and second frontal govern movements of eyes and head. These centres overlap one another.

These convolutions probably also receive sensory impressions from the skin, muscle, and very likely from the viscera, and thus

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[1] See Some Points on Intracranial Neoplasms considered from the Neuronic Standpoint, by the author. Read before the Section on Medicine, New York Academy of Medicine; Philadelphia Medical Journal, November 16, 1901.

are the cortical sensory centres for *muscle* and *tactile sense*, the superior parietal lobule being probably the location where sensations from the muscles are received.

Irritative lesions in the motor cortex cause local spasms (Jacksonian epilepsy), or general convulsions. There may also be paræsthesia of the parts convulsed, due to vaso-motor paresis.
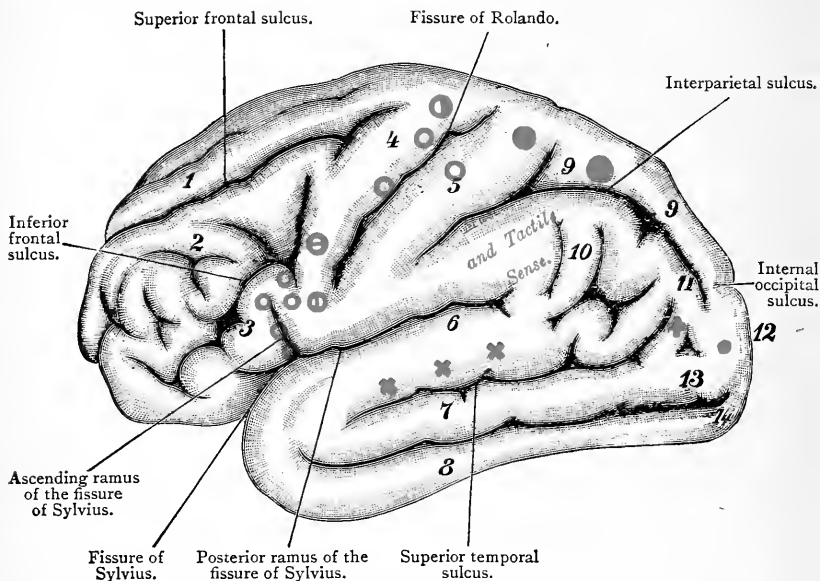
FIG. 41.—SCHEMATIC REPRESENTATION OF THE CEREBRAL CORTEX AND ITS CENTRES. (After Tillmanns.)

1. First.
2. Second.   } Frontal convolution.
3. Third.

4. Anterior.   } Central convolution.
5. Posterior.

6. Upper.
7. Middle.   } Temporal convolution.
8. Lower.

9. Upper.   } Parietal convolution.
10. Lower.

11. Gyrus angularis.

12. Upper.
13. Middle.   } Occipital convolution.
14. Lower.

In 4 and 5 on both sides of the fissure of Rolando, motor area for the upper extremity.

Motor area partly for the upper and partly for the lower extremity (great toe).

Motor area for the lower extremity.

Cortical area for the hypoglossal nerve.

Cortical area for the facial nerve.

(3) Motor aphasia.

(6) Sensory (auditory) aphasia with word-deafness.

(11) Aphasia with word-blindness.

(12) Region of the visual area (see also Fig. 10).

Destructive lesions.involving these same central motor convolutions cause paralysis of the muscles of the opposite side of body,

which soon become spastic. The paralysis is not always complete, since only a few centres may be involved. Muscles acting together (as of respiration) or those which express emotion (as the facial),
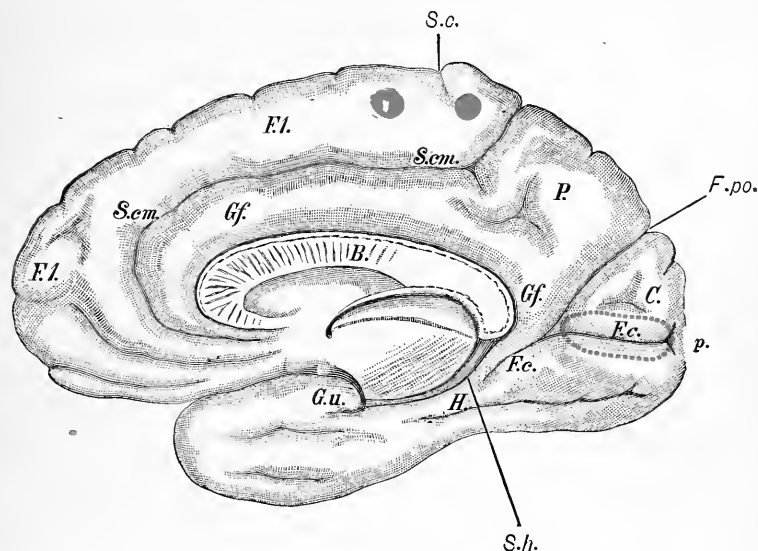
FIG. 42.—VIEW OF THE RIGHT CEREBRAL HEMISPHERE FROM THE MEDIAN SIDE.

B, corpus callosum divided longitudinally; *G. f.*, gyrus fornicatus; *H*, gyrus hippocampi; *S.h.*, sulcus hippocampi; *G. u.*, gyrus uncinatus; *S. c. m.*, sulcus calloso-marginalis; *F. 1*, first frontal convolution; *S. c.*, termination of the fissure of Rolando; in front the anterior central convolution with the motor area partly for the upper and partly for the lower extremity, and behind the posterior central convolution with the motor area for the lower extremity; *P*, præcunens; *C*, cuneus; *F. po.*, parieto-occipital sulcus; *p*, polus; *F. c.*, calcarine fissure; in the posterior part of this the visual area is shown by a red dotted line. (After Tillmanns.)

being represented on both sides of the cerebrum, are not completely palsied. Asteriognosis, or disturbance of sensation, may be found if the lesion extends to the posterior part of the parietal lobe.

The cells of the *ascending frontal* or precentral convolution, the *ascending parietal* or post-central convolution, the *superior parietal lobule*, the upper portions of the *first* and *second frontal convolutions*, part of the *cuneus*, the *paracentral lobule*, all control or originate voluntary motor impulses. These same centres also receive sensory impressions; hence this region of the brain is designated the *sensorio-motor* region. The trunk muscles and

leg muscles are largely represented in the upper portions of the precentral and post-central convolutions. The muscles of the arm have their centres in the middle third of the precentral and post-central convolutions; the face, tongue, larynx, and pharynx being represented in the lower part of these same *gyri*.

The Prefrontal Region.—This consists of the part of the frontal lobes in the anterior part of the third and ascending frontal convolutions. The centres that have to do with higher mental faculties (higher psychical centres) lie here—viz., those of judgment, reason, memory, attention, and comparison. It is the great association centre (Bianchi). It also is supposed to be a centre for the trunk muscles, according to Munk.

Centrum Ovale.—Lesions here may involve *association, sensory,* or *motor* fibres. A subcortical lesion may simulate a cortical palsy, but there is not likely to be spasm at any time except after invasion of the cortex. Hence, spasm always *follows* paralysis, just the opposite to that in cortical lesions. When the lesion is near the internal capsule the palsy resembles that due to lesion of it. There may be hemianopsia, hemianæsthesia, and if lesion is on the left side, aphasia in disease of the centrum ovale.

Corpus Callosum.—Symptoms are of partial or complete palsy, a hemiplegia gradually extending to the opposite side, with later development of dysphagia, dysarthria, and an insidious dementia. A double cortical lesion may, however, closely simulate symptoms of disease of the corpus callosum, but the greater asymmetry in the degree of muscle weakness would be in favour of cortical disease.

Corpus Striatum.—The function of this ganglia is not definitely known. It is very likely that, as excision experiments on dogs lead to suspicion of (and in a case under the care of Dr. John M. Swan and the writer, one of Friedreich's disease with temperature, which seemed to point to its degeneration as a cause of the pyrexia) the existence of a centre for heat, regulation is in the striatum.

Optic Thalamus.—This important ganglia is joined with the cortex of the frontal, temporal, parietal, and occipital lobes. It is the site of the ultimate ending of the optic tracts as well as of some sensory fibre neurones, which latter extend through their axons to the cortex. Very likely the main centre of regulation of body heat is in the thalamus. With the *external geniculate*

body and *corpora quadrigemina* the posterior part of the thalamus forms the *primary optic centre.*

The *corpora quadrigemina,* with the other two bodies just enumerated, form the *primary visual centres.* The remaining part of these centres control the reflex movements of the iris and ciliary muscles. The posterior bodies also, together with the internal geniculate bodies, are joined with the cerebellum and auditory centres, hence control in part *sensations of space* and *audition.*

Internal Capsule.—The motor and sensory projection fibres (or peduncular fibres) pass within very narrow compass through the internal capsule. In the region of the " knee " are fibres which pass from the cortex to the motor nerves of the eye, to facial and hypoglossal nerves, also those fibres which pass to the nuclei of the nerves that govern the other muscles that have function in speech, which are supplied by the vagus. The pyramidal tract fibres lie next posteriorly, those of the leg being also posterior to the arm. Posterior to all of these are the fibres that transmit common sensation and special sense impulses (hearing, vision, taste, smell).

Fig. 43.—Diagram of Motor and Sensory Representation in the Internal Capsule.

NL., Lenticular nucleus. NC., Caudate nucleus. THO., Optic thalamus. The motor paths are red and black, the sensory are blue. (From Osler's Practice of Medicine.)

As the pyramidal tract fibres are in a very compact mass in the capsule, a lesion here produces hemiplegia on the opposite side of the body, monoplegia being never produced by such a located lesion. If the lesion is in the posterior part of the capsule hemianæsthesia will result, associated or not with homonymous hemianopsia, and with muscular paresis.

Crus Cerebri.—A lesion of the crus will cause spastic palsy or paralysis upon the opposite side of the body, with palsy of muscles supplied by the motor oculi, since this nerve makes its superficial exit from the crus. The optic tract, as it crosses the

crus, may also be affected, producing homonymous hemianopsia. The *tegmentum* may, however, be involved without disease of the fibres of the crus, in which instance disturbance of common and muscular sensibility may occur associated with ataxia of the opposite side of the body.

The Pons Varolii or Bulb.—The gray matter here consists of cells which form nuclei of the peripheral neurones of the motor division of the fifth; also the sixth and seventh nerves. The pons is the centre for the ascending or sensory nucleus of the fifth nerve; also for the sensory and motor tracts on their way to and from the cortex. There are also neurones in the pons which connect with the cerebral and cerebellar cortices.

Cerebellum.—Here the gray ganglionic masses are in the centre, but the *gray matter* proper is upon the cortex. The *vermis* or middle lobe is alone known positively as to function, and is joined with sensory and motor paths of both brain and cord, the auditory nerve, some of the nuclei in the pons, and with the inferior olivary bodies of the medulla. The *functions* of the cerebellum are not definitely ascertained in detail. It is known to take part in maintaining the equilibrium of the body, in muscular movements, and in maintaining our relations to surrounding objects. Hughlings-Jackson claims that disease of the cerebellum causes palsy of the trunkal muscles also, and that the cerebellum maintains muscle tone.

Medulla Oblongata.—The lower motor neurones or nuclei of the eighth to twelfth cranial nerves lie here, except the spinal root's centre of the eleventh, which is in the cord. The oblongata is of course only the expanded portion of the cord, and the cells lying here have the same sort of function as do those in the gray matter of the cord.

In the medulla are also found reflex and automatic centres controlling the respiratory and circulatory functions, secretion, and the visual movements. The olivary bodies (superior and inferior) are small masses of gray matter in the medulla, and are joined with the basal ganglia, cerebellum, and cord, having to do in part with co-ordination and equilibrium.

A lesion high up in the oblongata may damage the pyramidal tract above its decussation and the hypoglossal nerve upon the same side as the lesion, producing an *alternate* paralysis.

Affection of the nuclei gives rise to the so-called bulbar symp-

toms—those of glosso-labio-pharyngeal paralysis. Death may follow suddenly from involvement of the respiratory or cardiac centres. Polyuria or glycosuria may also result from chronic lesions of the medulla.

The functions of the OCCIPITAL LOBE have been mentioned under diseases of the optic tract.

Cortical centres concerned in speech are to be described next under Aphasia.

APHASIA

By this we mean disturbance of the power of communication by the ordinary signs of language. The word aphasia, which signifies loss of speech, is the general designation applied to all forms of defects of the elements of speech due to disease of the cerebral hemispheres. This is usually a lesion involving the cortical centres of the speech mechanism. Aphasia is to be distinguished from disturbance of speech that follows mechanical trouble with articulation due to lesion of peripheral organs or nerves, and also from speech defects due to the lesion of the cranial nuclei or of the association and co-ordination tracts of speech in the oblongata, pons, and cerebellum. Aphasia is, therefore, limited to a partial or complete loss of the power of comprehension or expression of language. It is divided into two classes—*motor* and *sensory* aphasia. The former may be designated emissive, and the latter a receptive type. Of the motor form we have subdivisions considered as elementary—namely, (1) aphasia, or complete motor aphasia, (2) agraphia, or inability to write.

Of the sensory form we have (1) word-deafness, or auditory aphasia, and (2) word-blindness, or visual aphasia. Mills designates another form of elementary aphasia—namely, verbal, or that variety which is due to the lesions of the naming centre. Apraxia is the form where the patient is not able to understand the uses of objects.

Motor Aphasia.—Aphemia, as Brocker, Ross, and others have designated motor aphasia, is divided by some into two or three forms, the *first* being strictly limited to that produced by lesion of the foot of the left third frontal convolution, the so-called Broca convolution. Broadbent names *another* variety, in which the lesion is at the foot of the second central convolution back of the Broca centre. Total destruction of Broca's convolution in right-handed

people makes spoken speech impossible for a time at least. Later, the hitherto untrained centres in the right third frontal convolution develop. The patient may thus acquire or regain partially the faculty of speech. Usually, however, recovery in part is due to the fact that the centre was not entirely destroyed. The reason for the speech centre being on the left side is, theoretically, assumed to be that this side is much more highly developed than the other, in obedience to the principle of the economizing energy, most people being also right-handed. *Articulative ataxia* is nothing more than the old *ataxic aphasia*, so-called, or the *asynergia verbalis* of Lordat—i. e., the existence of incomplete motor aphasia, which is
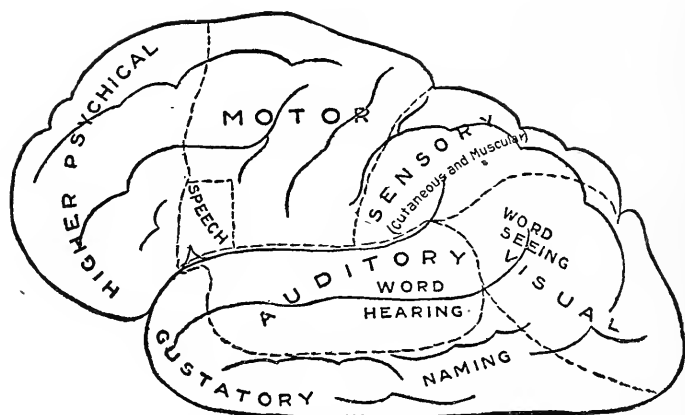
Fig. 44.—Zones and Centres. External Aspect. Cerebrum. (After Mills.)

generally the sequel of the complete form. Most cases of motor aphasia also suffer from *agraphia*. This loss or defect may be due to lesions of the centre for word-seeing, which centre is placed at the caudal extremity at the second or medio-frontal convolution, and in close relationship with the upper or mesial boundary of the true speech centre. True agraphia is an almost invariable result of destruction of Broca's centre, however. It is probable, as Wiley concludes, that motor agraphia, as far as writing with the right hand is concerned, may result from a lesion in the graphic centre in the posterior portion of the second frontal convolution.

Pantomime study is only second in importance to that of spoken speech in the consideration of aphasia. It is in general a part of speech, and so closely associated that disturbance of

speech is always accompanied by disturbance more or less of pantomime.

Amemia is loss or impairment of power of expression by signs, when due to cerebral disease.

Paromemia is confusion of the signs in efforts at expression through them.

Sensory or Receptive Aphasia.—This is considered usually as having three special forms: (1) auditory aphasia, or word-deafness; (2) visual aphasia, or word-blindness; (3) and apraxia, also called mind-blindness, soul-blindness, and object-blindness. Physiological investigations, including those of Ferrier, prove (1) *word-deafness* is due to lesion in the posterior third or first and second temporal convolutions, as verified by Mills, who contends " that it is not complete unless the corresponding region on the right hemisphere is also impaired or destroyed." Other symptoms present in this form are inability to read aloud correctly. The patient is unable to verify what he reads by hearing. In the complete form he cannot echo spoken words. Paraphasia and paragraphia may also be present in these cases, as may also verbal amnesia, and as may articulative amnesia. But it is probable that in some of these cases the amnesic phenomena are due to the involvement of the centre, or of tracts leading from the centre of word-hearing to the conceptional or motor centres. Music-deafness may be associated with word-deafness. But from the fact that it is sometimes present when auditory aphasia is absent, it is assumed that in some people the faculty for music belongs in both hemispheres of the brain to a greater degree than does the faculty of hearing. Between the auditory centres at the base of the brain and the cortical auditory centres in the left temporal lobe are also entering tracts for hearing. A lesion of these tracts, which probably go from both sides of the brain, will give rise to a form of word-deafness. Lichtheim has placed the entering auditory tracts chiefly in the left temporal lobe. A lesion of this tract will cause outer word-deafness. A case of lesion restricted to this entering tract alone has been reported by Lichtheim. We must have entire comprehension of cortical anatomy aphasia and complete understanding of the auditory path before this matter becomes clear.

(2) *Visual localization*, like auditory localization, has also certain tracts to be considered from an anatomical and physiological point of view before we can understand amblyopia, hemianopsia,

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etc. Henschen believes that the centre for the macular field lies
in the calcarine cortex. There is also doubtless stored near the
visual centre images of words, letters, and probably of objects.
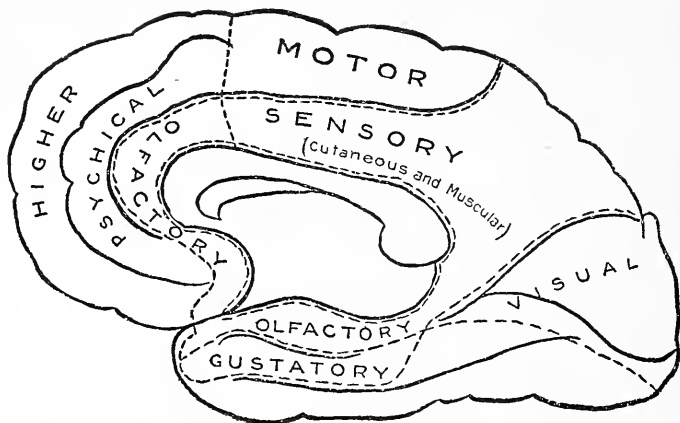
Fig. 45.—Zones and Centres. Mesal Aspect. Cerebrum. (After Mills.)

This is likely in the angulo-occipital region or on the lateral
surface of the hemisphere, as located by Ferrier. Partial destruc-
tion will produce word- or letter-blindness.

Alexia, or the inability to read, will, of course, be produced
by such a lesion as will cause agraphia so far as this is dependent
upon sight. Patients who have been rendered *alexic* or *word-blind*
(lesion of centre for word-seeing) can sometimes write their names
or simple words; apparently doing so through touch or by recog-
nition of psycho-motor images. Between the primary optic cen-
tres at the base of the brain and the cortical centres recited are
certain entering tracts for vision (optic radiations of Grateolet).
It must be remembered that the primary cortical visual centres are
connected with half of the retina on each side—the same half on
which the centre lies—the fibres passing to the angular region in
each hemisphere. This is the centre of eye functions. A lesion
which severs the tracts coming from both occipital lobes to higher
centres will cause word-blindness, but not agraphia. Commonly
such a lesion will also produce hemianopsia, since the radiations
of Grateolet are usually involved.

(3) Not infrequently associated with word-blindness is another
disorder, which has been variously called *mind-blindness,* soul-
blindness, and object-blindness. In examining for this, the physi-

cian tries to determine if the patient shows signs of recognition of various objects. He may not comprehend the use of the simplest things and he may not recognise intimate friends, excepting by touch or by hearing their voices. (Occasionally there is added to this form a *psychic deafness* in which the patient cannot recognise a friend by the sound of the voice.) The centre for mind-blindness may or may not be separate from the centre for visual images of words (visual aphasia). Both hemispheres of the cerebrum doubtless take part in the storing of images in equal degrees. The mental percepts of objects and of names are the results of definite processes of cerebration (Mills). *Ideation* is particularly disturbed through lesion of the radiating fibres, whereas the determination of names is disturbed through lesion of the centres for percepts only.

Mills, with Broadbent, holds the view that a naming centre exists in the cortex of the brain. Destruction of this centre will produce *verbal amnesia*. A form of verbal amnesia, called *articulative amnesia,* would be due to partial destruction of this centre.

Conducting aphasias are those forms of speech disturbance
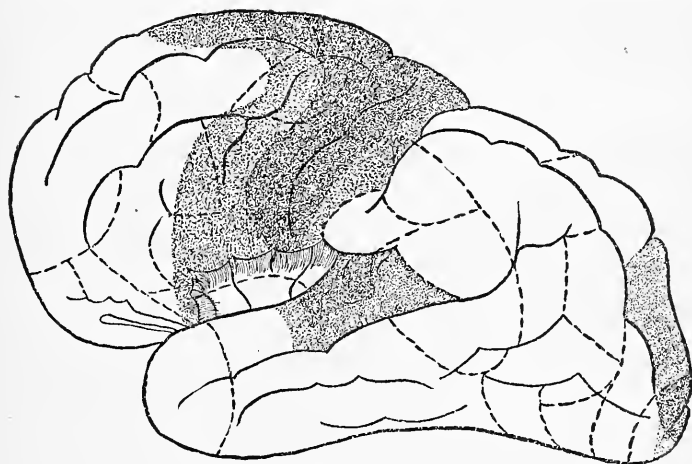
FIG. 46.—FLECHSIG'S ASSOCIATION AREAS—LATERAL ASPECT.

which are due to defects or destruction of the tracts associating various regions concerned in the mechanism of speech. Some of these are *paraphasia; paragraphia,* the misuse of words in writing; *paramemia,* the misuse of signs; *paralexia,* misuse in reading of

syllables or words; and *dyslexia,* which is difficulty or fatigue shown in reading.

Examination for Aphasia.—First: determine associated phenomena of aphasia, as gesture language, already detailed. Test for these carefully. Second: extra-graphic symbols should be sought out, such as the methods of implying numbers, algebraic signs, etc. Third: the determination of mind-blindness. Fourth: emotional and intellectual faculties, which include determination of emotion, mentality, etc. Fifth: motor and sensory functions should be studied. These should all be critically observed.
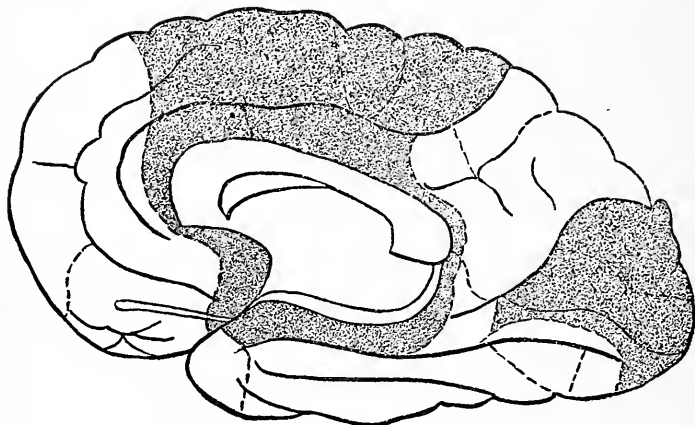
FIG. 47.—FLECHSIG'S ASSOCIATION AREAS—MESIAL ASPECT.

The definite subheadings, as given by Mills, are as follows:

The methods of (A) *spoken speech* should be determined—

1. Inquiry as to how the patient receives and interprets.

2. (*a*) How is it produced? making a record of words, etc. (*b*) Evidence of amnesia and its companionable symptoms—articulative amnesia and paraphasia.

3. How is it repeated or echoed?

(B) *Written Speech*—

1. How is it received and interpreted?

2. Is sight good, is hemianopsia present? etc.

3. How is it produced?

4. Ask the patient to write his name.

5. How does the patient write to dictation or from copy? Try him in both ways, and if he is able to do either or both, try to ascertain if he understands the meaning of what he writes.

CHAPTER XI

OTHER DISEASES OF THE BRAIN AND MEMBRANES

GENERAL SYMPTOMATOLOGY

THERE are certain general signs and symptoms that are found more or less constant in almost all organic diseases of the encephalon. These symptoms may be placed in five classes: (1) general symptoms of brain irritation; (2) general symptoms of brain pressure; (3) symptoms of focal irritation or destruction; (4) symptoms due to local pressure; and (5) those due to the pathologic process itself.

The symptoms of brain irritation are headache, vomiting, vertigo, photophobia, mental irritability, insomnia, a sense of pressure and fulness about the head, noises in the ears (tinnitus) or in the head, tenderness of the scalp, and in bad cases convulsions, paralysis, or delirium and stupor.

Brain *compression* has as symptoms headache, vomiting, mental hebetude or dulness; or it may be some form of paralysis, contraction of pupils, and finally coma. Retraction of abdomen (scaphoid) accompanied by constipation is often present.

Symptoms of brain compression may be associated with anæmia, œdema, or increased blood pressure; or, indeed, in states of malnutrition, when the brain mass is impoverished, symptoms of compression may prevail. Irritation and pressure symptoms may interdigitate, so that at times it is impossible to differentiate one from the other. Encephalic *irritation* symptoms are usually associated with hyperæmia.

Focal or *local* symptoms depend almost absolutely upon the location of the lesion causing them. If the motor area is affected, twitching, spasm, or convulsion would be likely to follow early; while when destruction at the same site occurs, the symptoms will be of paralysis. If in a sensory centre or tract, the first symptom would be of paræsthesia or hyperæsthesia; the later one being of anæsthesia of the part supplied by the centre involved.

Symptoms due directly to the pathologic lesion itself may be meagre; just as in tumour of the spinal cord, for example, the symptoms of irritation, pressure, or even destruction or local disturbance of circulation may be dwarfed by septic infection.   General symptoms, the result of septicæmia, such as chill, fever, sweating, etc., may become paramount.

Hemiplegia and *aphasia* are symptoms that may be caused by so many different kinds of lesions that they demand some special study here.   Both groups of symptoms pertain in general to motor or sensori-motor disturbance.

Hemiplegia is paralysis of one half of the body, as a rule involving the side opposite the lesion.   The arm is usually the most palsied, the leg next, and the face least.   Hemiplegia may be acute or of slow onset, the former being due to hæmorrhages, cerebral softening, or more rarely to injuries or to inflammations.   The latter is usually very insidious, and is generally caused by tumours or slowly developing areas of sclerosis; but this may be only a part of diffuse sclerosis.   The particulars of hemiplegia will be given under Special Diseases of the Brain.

MALFORMATIONS OF THE BRAIN AND ITS MEMBRANES

There is little practical importance to this subject as regards congenital malformations, since in most cases the monsters die—a most fortunate result.   The following are some of the types:

Brain.—Anencephaly; micrencephaly and microcephaly; porencephaly; malformations or absences (cyclopia).

Brain and Membranes.—Acrania; meningocele; encephalocele; hydrencephalocele.

With acrania, anencephaly is always present.   In anencephaly, the cerebellum and part of the basal ganglia existing may permit the child to live for a short time after birth.   In micrencephaly the brain is but partly developed; and if in addition the cranium is also abnormally small, as is generally so, the name microcephaly is given.   This is due to lack of growth of the brain, and probably not at all to premature growing together of the cranial bones, as once contended by Virchow.   If the circumference of an adult cranium is less than 42 centimetres it will contain a micrencephalic brain.   Normally the weight of an adult man's brain is 960

grammes, while 880 grammes is the weight of an adult woman's brain. It is 14 per cent of body weight at birth and 2.4 per cent at adult life. Porencephaly is often the result of hæmorrhage. In cyclopia there is an undivided anterior cerebral vesicle, and the orbits form a continuous cavity with a single rudimentary eye. Meningocele is a hernia of the brain membranes. In encephalocele the brain also protrudes through the cleft in the skull.

FIG. 48.—LACK OF DEVELOPMENT. LEFT HALF CEREBRUM. (Specimen from woman at the Philadelphia Hospital.)

These forms usually occur in the occipital region and in the median line. In hydrencephalocele there is a sac with fluid contents.

MENINGOCELE

This may be either of the membranes of the brain or of the cord. It consists in the protrusion of the meninges out of the natural position within the skull or spinal cord. In the case of the brain, where there is also an existent internal hydrocephalus, the distended ventricle may form a part of the tumour, in which case

it is then designated a hydrencephalocele. Usually such tumours are covered with the skin of the scalp. At other times they are devoid of the cutaneous covering. The most frequent position for the appearance of these tumours is in the anterior portion of the skull, very seldom occurring at the base of the brain. This is largely on account of the thinness of bone and the position of the fontanelle openings. The hernia is *caused* by increased cerebral pressure from within, due to excessive fluid, and frequently to teratological defects as the primary cause. Adhesion of the amnion to the cephalic end of the embryo is probably an important factor in the production of the *congenital* condition type. Disease of the meninges in early life may be the cause of some *acquired* cases. Sex has no influence in their production. Maternal impressions and fright are said to be exciting causes of these malformations. Usually the length of life in infants affected with this deformity does not extend beyond one year. Cases have been reported, however, where they reached adult life.

Symptoms.—Membranes protrude. Drowsiness and mental enfeeblement, also paresis and convulsions, are the most important symptoms.

Pathology.—Usually the condition is an embryological defect. If disease of the membranes has been the primary cause, the pathological condition or cause will be that of the primary disease, such as meningitis, acquired hydrocephalus, etc.

Treatment.—This is most uncertain. If the protruding mass is small it may either be excised by the surgeon or compressed into the cranial vault. Some authorities have advised the withdrawal of the fluid in the sac; and where the membrane extruded is quite isolated from that within the skull, injection of carbolic acid, with the hope of setting up subacute inflammation, which may shrivel the mass in question, can be tried.

APOPLEXY (CEREBRAL HÆMORRHAGE)

By this term is generally understood hæmorrhage into some part of the brain, and in the majority of instances the bleeding is from the lenticulo-striate arteries. These supply nutriment to the motor cortex and important ganglia at the base of the brain, from which projectile fibres extend to the spinal cord and transfer nerve energy to the lower neurons and extremities.

Apoplexy is sometimes called a " stroke," or " paralysis "; and while paralysis is the usual result of a hæmorrhage into the central nervous system, it is more exact to speak of apoplexy than any of the other terms commonly used by the laity. The physician understands a broader definition of apoplexy even than that above given —i. e., *thrombosis* of cerebral blood-vessels, or *embolism*, either, though more limited, practically produces similiar signs and symptoms; the former insidious, the latter sudden. As the treatment of these three conditions would not much vary, it will be
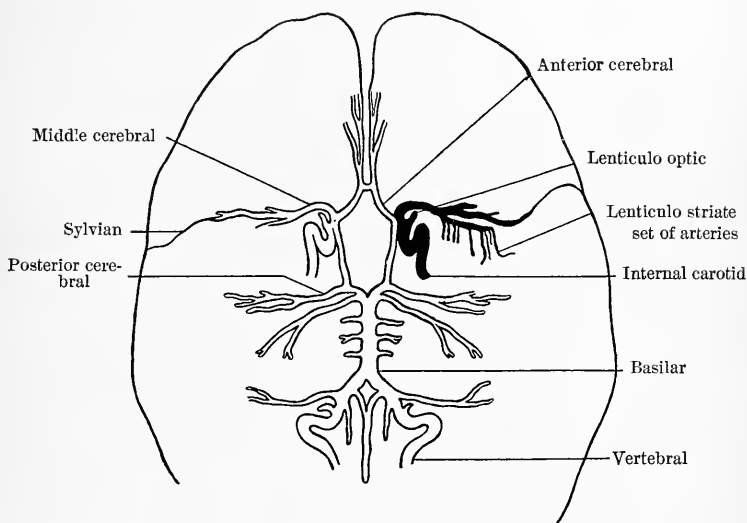
Fig. 49.—Showing Arteries at the Base of the Brain. One, the lenticulo-striate, is called the artery of cerebral hæmorrhage. (Slightly modified from Dercum.)

unnecessary to more than mention them. The predisposing causes of true apoplexy are in the following order of occurrence: *first,* chronic alcoholism; *second,* syphilis; *third,* other infectious diseases. The predisposition to cerebral hæmorrhage with what might be called normal sclerosing or hardening of the vessel walls due to advancing years, must be considered; and here it is especially that heredity plays an important rôle in *etiology.* In proportion, then, as persons past middle life develop arteriosclerosis, will the tendency to this serious malady occur. Induced plethora from overeating is a predisposing cause. Physical and mental strain are exciting causes. Night or early morning hours are the

most favourable times for cerebral hæmorrhage, and especially for embolism and thrombosis. Singularly, it often happens when the patient has been feeling particularly " well," although it will be found there have been some days or months of sense of vertigo with fleeting attacks of congestion of the brain. The attack may appear in such a predisposed individual following exertion, straining at stool, or after partaking of a hearty meal.

Signs of Attack.—Rather sudden loss of consciousness preceded by thickness of speech, or motor aphasia, accompanied with flushing of the face, extreme objective vertigo, followed by falling
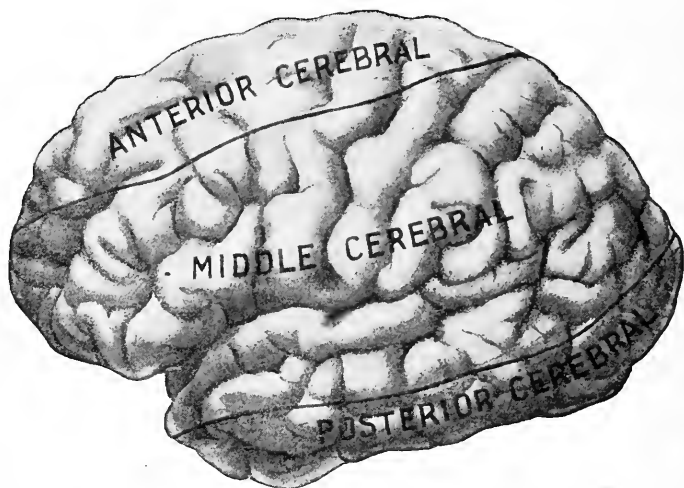
Fig. 50—Showing the Portions of the Cerebral Hemispheres Supplied by the Anterior, Middle, and Posterior Cerebral Arteries. (Redrawn from Dana.)

in unconsciousness if the patient is standing or sitting. With this there is more or less stertorous breathing, and perhaps clonic convulsions will precede the ultimate paralysis, which is usually of one side of the body, owing to the location of the hæmorrhage on the opposite side of the cerebrum. It will be found within a half hour that the temperature of the patient has risen slightly, the coma becomes more profound, following the marked restlessness which is seen just after the stroke. There is slight hyperæsthesia of the paralyzed side, as shown in a few cases where the coma is not too profound. The increase of temperature is more pronounced on the affected side, and averages half a degree by the axillary record

or less by surface thermometers. Hyperpyrexia may rarely occur, and is always an unfavourable, usually a fatal sign (Fig. 52).
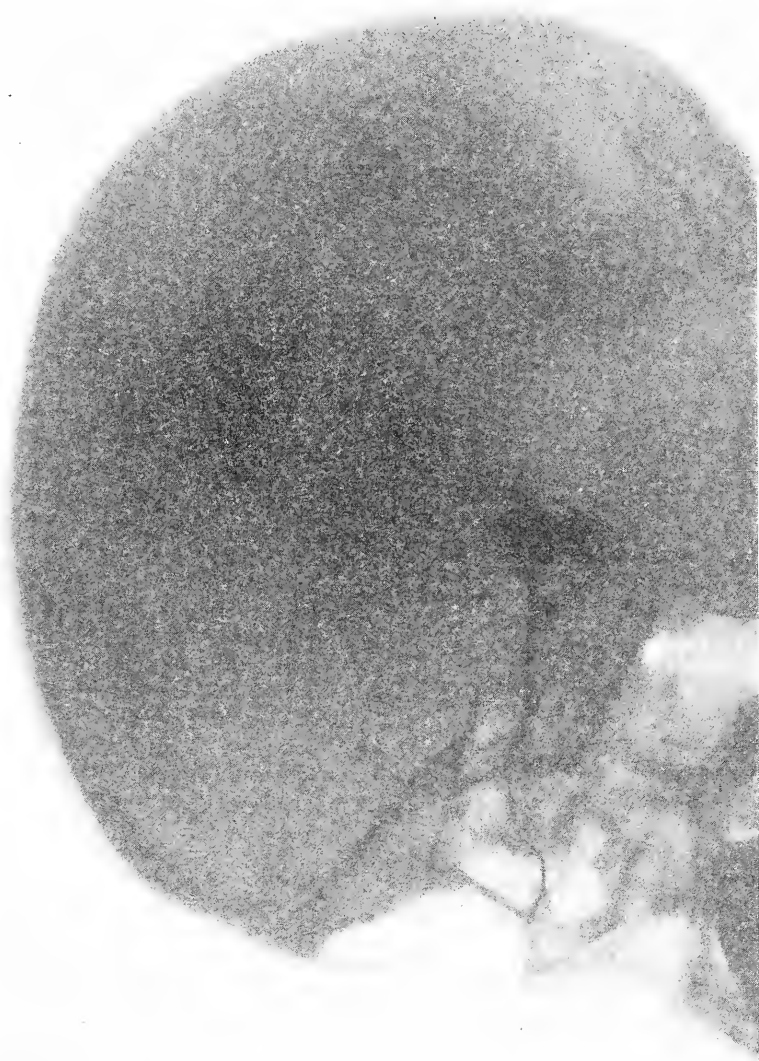
FIG. 51.—X-RAY PHOTOGRAPH. Shadow showing area of thrombosis and hæmorrhage motor area cerebrum.

The paralyzed extremities when raised from the couch will drop flail-like; the patient occasionally, on partial recovery, will, however, be able to move the other side of the body. There may also

be retention of urine, which must be guarded against by the nurse. The deep reflexes on the paralyzed side are always diminished or absent in this stage. *Conjugate deviation* of the eyes, or a turning of the eyes away from the paralyzed side in cerebral lesions, may exist; or the eyes may remain "fixed." If convulsions occur the "deviation" of head and eyes may be *towards* the palsied side.

At the end of three or four hours, if the case does not go to a fatal issue, the patient begins to improve. The coma now becomes less profound, the reflexes appear again in the paralyzed side, the patient may recover power in muscles, perhaps an extremity, and he may turn from the supine position in which he lay to one or the other side. Here the nurse should be instructed as to care to prevent pressure paralysis, which may be caused by lying upon the enfeebled arm. The *chronic* stage now sets in. Gradual restoration of speech takes place in the majority of cases, although there may remain some form of dysarthria, as "thickness" of speech, for some months in even the most promising cases for large recovery of power. There remains difficulty in swallowing, and the saliva may dribble from the mouth for some months. As a rule, the tongue is protruded towards the paralyzed side and the face drawn towards the sound side, although there is no disturbance of taste.

The muscles of the paralyzed side do not degenerate in the sense of showing "reaction of degeneration" by the galvanic current, and there is rarely true trophic wasting,-only that due to disuse of the affected members. In the average case, at the end of a few weeks, the patient will be able to move about with the assistance of crutches or cane. The manner of progression will be typically characteristic of the so-called hemiplegic gait. The affected limb is thrown out and forward, describing the arc of a circle, and then the foot is dropped down flail-like upon the ground. This peculiarity in walking is caused by the patient throwing his trunk forward and to the sound side, due to the fact that the extremity being paralyzed, compensatory movements and muscular effort must be made by the other limb. Substituting this flaccid hemiplegic gait, in the course of a few weeks there develops in the patient spasticity of the muscles of the affected side; and then the paralyzed member will be, with the partially regained power, pushed along in a spastic fashion, the toe being "dug" into the ground.

Fig. 52.—Hyperpyrexia in Cerebral Haemorrhage; Male, aged Sixty-one Years.
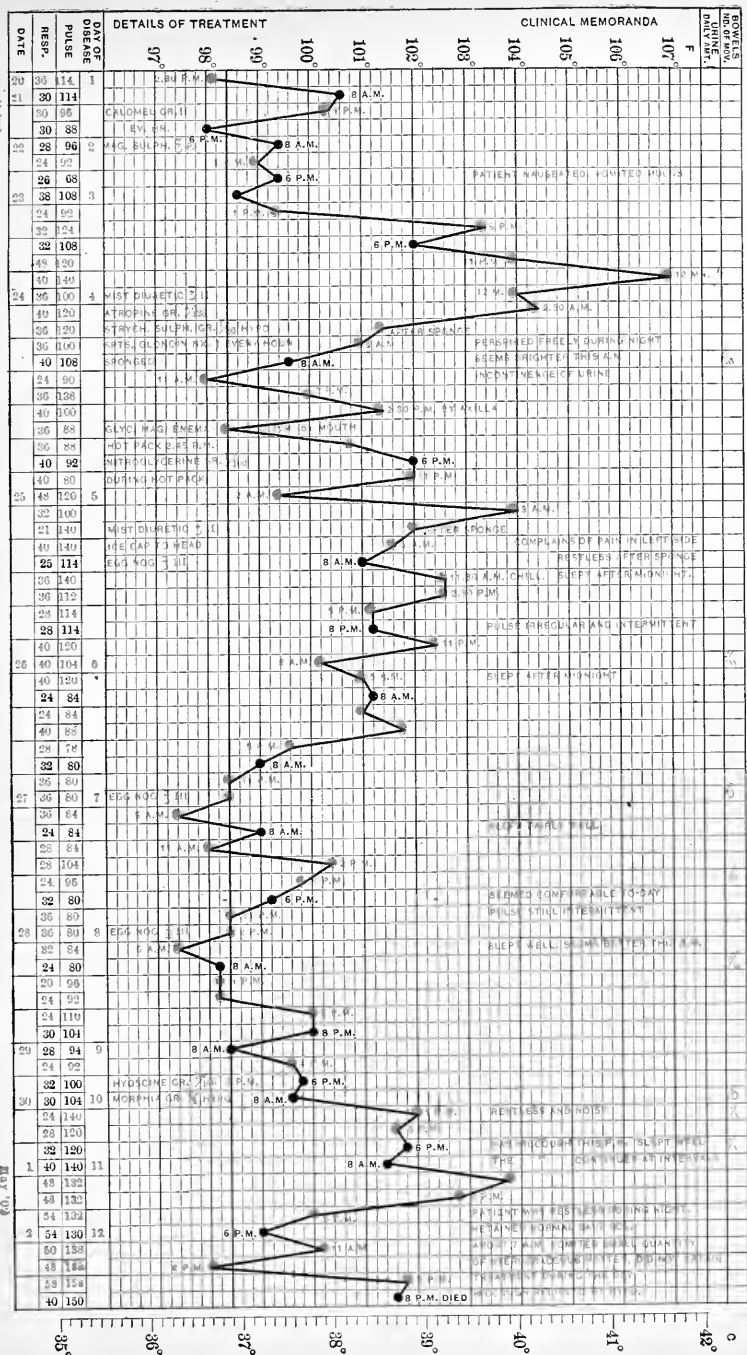
The affected arm, which has thus far hung flail-like, improves more slowly as to power than does the lower limb; but with the return of power, flexion of the elbows and fingers takes place. This is due to secondary contraction of the flexor muscles, which are disproportionately increased in power over the extensor group of muscles. In the course of the affection there will be found secondary degeneration, and this may extend to the opposite side of the spinal cord, causing great increase of the deep reflexes on both sides of the body. As the case progresses, joint symptoms are not unusual; the disuse of the joint and lowered trophic condition sets up a low grade arthritis, the affected joint becomes painful and semi-ankylosed. Subacute neuritis in the affected extremity may also be set up. Great wasting of the extremity is the exception. There is never reaction of degeneration in the paralyzed muscles in cerebral lesions, as already referred to in a previous paragraph. Sweating upon the paretic side of the body is not a usual symptom, but when present shows vaso-motor weakness. The following hygroscopic observation has been made for me by Dr. H. E. Wetherill:

Case of M. K., aged thirty years, female: Right palm, 82.5 per cent; left palm, 96.0 per cent; abdomen, 2 inches above umbilicus, 84 per cent; mid-scapular region, Wurtzer's uncovered instrument, 70 per cent; temperature by the mouth, 98.3° F.; temperature of the ward, corrected, 72° F.; relative humidity of air of ward, 84 per cent; barometric pressure, corrected, 30.045 inches.

Result in this one case: "It would appear that the skin moisture is increased in the diagnosed condition of left hemiplegia, notwithstanding it was a damp day."

Pathology.—The hæmorrhage is generally due to disease of the blood-vessels, which is usually a degenerative arteritis:

First, attacking the media. *Miliary aneurysms* form. The aneurysms occur most frequently upon the cortical vessels, and may also extend to the smallest arterioles. On section of the brain-substance they appear as localized, dark bodies about the size of a pin's head.

Second: *Endarteritis* and *periarteritis* may lead to apoplexy, either by producing miliary aneurysms or coarse ones of the larger vessels, as of the circle of Willis.

Third: *Fatty degeneration* of the small vessels occurring in purpura, scurvy, leucocythæmia, marasmic conditions, and acute

infectious diseases. Atheroma is usually found in the larger vessels.

The parts affected in hæmorrhage, in the order of frequency, are the caudate and lenticular nuclei, meninges and cortex, centrum ovale, optic thalamus, pons, cerebellum, and medulla. Ventricular hæmorrhages are usually secondary to hæmorrhage into the neighbourhood of the basal ganglia.

After a hæmorrhage there is first coagulation of the blood, which soon begins to soften and be absorbed. The inflammation occurring about the clot usually causes the formation of a fibrinous wall around it, which forms a cyst with fluid contents. In other cases, instead of the formation of a cyst, there is proliferation of connective tissue and the formation of a pigmented scar.

Secondary degenerations follow, due to the cutting off of nerve-fibres from their parent cells, which in most cases, as hæmorrhage in the region of the internal capsule is very common, would involve the pyramidal tract and be the cause of late rigidity and increased reflexes (the inhibitory influence of the cortical cells being cut off).

Diagnosis.—The history of an apoplectic stroke with consequent hemiplegia is the important point. *Embolic apoplexy* is to be diagnosed largely by history of cardiac disease and in the finding of a well-marked organic heart disease, as shown by murmur over the præcordia. In embolic apoplexy, too, there is not so constantly disturbance of consciousness, and the sequent paralysis is very apt to be more permanent and localized. Following the embolic form also, careful examination may reveal alteration in the physical signs of the existent heart disease; for instance, the murmur may partially disappear, or at least change in quality very materially. In *thrombosis* there is no positive way of telling, except by noting previous history of thrombosis in other parts of the body and by extensive arterial degeneration: also of a slow onset.

Prognosis.—About 50 per cent of cases of apoplexy recover from the attack; 75 per cent of these are very apt to have recurrence; the remainder go on through life with motor paralysis of the parts involved, life being perhaps curtailed, on an average, by a decade. The expectancy of life in these cases, therefore, is relatively proportionate to the heredity of longevity.

Treatment and Nursing.—In the apoplectic attack the patient should be guarded very carefully, placed in the supine position,

with the head partially elevated; ice-caps should be placed to the head and hot-water bottles to the feet, being careful not to burn the patient, since obtunded sensation by the unconsciousness would prevent the appreciation of heat. Friction to the lower extremities will aid in restoring circulation here, and thus favour the relief of congestion in the brain; or a mustard bath may be of value. A *denture* or other foreign body should be carefully sought for, since there would likely be strangulation from this source. It may be necessary in some cases to produce traction on the tongue, by means of a handkerchief or an artery forceps " clipped " on to the tip of the organ. Respiration can also be aided by gentle, firm elevation of the larynx with the tips of the thumb and index-finger. The inhalation of oxygen is a method I have nowhere seen used, but it is scientifically ideal for oxidation, and should be tried in serious cases when other methods have failed. The revulsant action through the intestinal tract should be brought about by a hydragogue cathartic. For this purpose croton oil is employed by the physician, 2 to 4 drops being placed upon the tongue directly or in solution upon sugar. The reason for this therapeutic measure is that the congestion of the mucous membrane causes great exudation of serum from the blood, and in consequence of such local and general depletion, the marked relief of the congestion in the brain. Venesection or bleeding by leeches are important adjuncts in sthenic cases. At the end of forty-eight hours small quantities of milk may be administered, although for some days there should never be excess of nutriment placed in the stomach. The patient should be kept warm, with especial attention given, however, to ventilation. Stimulants should be eschewed save in the use of strychnine, which may be prescribed for heart failure only.

In the " getting up " of the patient, as indicated before, exercise should be sparingly indulged. The time of sitting up (not before a fortnight) should be very gradually increased from day to day until there is a proper adjustment of the altered and hampered circulation. A patient generally has to employ crutches in his first endeavour at walking, and this should be encouraged until sufficient power has been regained, when the crutches can be substituted by a cane. When contractures occur in the paralyzed extremities, and even before this to prevent such deformities, it is wise to employ massage and stretching in the affected members.

The use of iodide of potash is the most valuable remedy at our command for the absorption of the exudate in the brain. We have seen some excellent results accomplished with this drug, especially in the specific cases where the remedy can be gradually pushed up to several hundred grains a day. Extra exertion, as in lifting, should be guarded against, the bowels must be kept regular, and the patient should live a life much less active than usual. With this outline, subjects of apoplexy may live many years in fairly comfortable health, although hampered considerably as to motion and mental capacity.

Cases of cardiac disease are always more serious, since life is menaced by the fact that a vegetation may be carried into the brain at any time.

ENCEPHALITIS

By this is meant an inflammation of the encephalon or brain mass.

Causes.—The causes of this disease are injuries, *infectious diseases and local infections;* or an *arterial sclerosis*, by cutting off nutrition to the cortex of the brain.

Varieties.—These are acute, as the hæmorrhagic; and secondary or chronic, which is divided into the septic (or brain abscess) and the simple chronic encephalitis, which is divided into *encephalitis superior,* where the third nuclei are affected, and *encephalitis inferior,* where the cranial nerves below the third are involved.

Symptoms of Acute Encephalitis.—The symptoms of acute hæmorrhagic encephalitis are mental confusion, slight rise of temperature, palsy of the extremities without actual paralysis, and but seldom convulsions. There may be palsy of the third nerves. The disease is very rapid in onset, and may be accompanied by paralysis of one side of the body (hemiplegia).

Prognosis.—This is bad, the disease lasting from a few days to a week or even ten days, the patient dying of pressure upon the brain or from involvement of the respiratory or cardiac centres.

Pathology.—This consists in an inflammation of the cortical cells of the brain with minute hæmorrhages scattered here and there throughout, and in some cases a distinct hæmorrhagic effusion may exist. There may be optic neuritis, but seldom choking of the disk.

Symptoms of Chronic Encephalitis.—Symptoms of chronic septic encephalitis are all the above symptoms in less severity, plus the addition of chills in some cases, and in few a rise of temperature, although depression of temperature is frequent even in cases of abscess of the brain. There is usually a history of ear disease or chronic nasal disease preceding the attack, and with this history the case (giving symptoms of inflammation of the brain) will be found to be of septic nature. In 90 per cent of all cases sepsis will be found.

There is very frequently localization of the inflammation in septic encephalitis, although there may be multiple foci scattered throughout the encephalon; if local, such symptoms as convulsions or paralysis may be set up by the irritation or destruction through the abscess. Choked disk is not the rule, although optic neuritis is likely to occur; and especially is this the case if the abscess is cerebellar in origin, which is the most frequent site of abscesses, because chronic middle-ear disease is the most frequent cause of brain abscess, and the cerebellum being adjacent to the ear would explain the prevalence in this locality. If the abscess is in the cerebellum, the cerebellar gait will be a feature in the diagnosis; if in the motor area, convulsions and paralysis would be looked for; if in the spheno-temporal lobe, deafness should be looked for; or if in the posterior parietal lobe, asteriognosis and mind-blindness may be a part of the symptomatology; whereas if located in the frontal lobe there are no special symptoms, the mental stupor, however, being profound.

Diagnosis.—Differentiation should be made from brain tumour. This is done by the history of the case and the development of irregular temperature in encephalitis and its more rapid progress.

Prognosis.—The growth of brain abscess is usually rapid, the patient dying within a few months after its development. It may, however, last for years, and become incarcerated and form a " cold " abscess, which may at any time, from trauma especially, develop symptoms of acute septic encephalitis and result in death.

Treatment.—This is surgical alone. If a case is diagnosed, the surgeon should be called into consultation immediately for decision as to operation. The best results are obtained in those that are cerebral rather than cerebellar, on account of the anatomical difficulties in the latter.

The symptoms of *chronic polio-encephalitis superior* are those of simple encephalitis of mild type. There is no pain connected with the disease, and the third nerve being involved, we have in addition ptosis, exophthalmos, and iridoplegia. The eyes are also held in external strabismus if the sixth nerve is still intact.

The symptoms of *polio-encephalitic inferior* are those of ordinary encephalitis, plus those of the involvement of the nuclei of the fourth and the cranial nerves below this. If the pathetic alone is involved there would be, in addition to the above symptoms, rotation of the eyes up and in. There is an absence of temperature in cases of chronic nature. According to the nerves involved below the fourth nerve, will we have symptoms of paresis of those nerves.

Prognosis is very unfavourable. Some amelioration may be expected under thorough treatment.

Diagnosis.—It should not be confused or confounded with any other form of disease, the particular point being preservation of consciousness with the incomplete palsies suggested above.

Treatment.—This is palliative; quiet is essential; apply cold to the head, and use supportive measures, such as strychnine and belladonna in small doses, or of nitroglycerine, to support the heart. Iodides should be used, as also sorbefacients where there are any symptoms indicating exudates about the inflammatory area. Individual galvanization of muscles of paralyzed parts, such as of the eye muscles, may be of some value. These patients may live for a long time—years; however, they are very likely to be carried off by an intercurrent affection, as pneumonia.

BRAIN TUMOUR OR CEREBRAL NEOPLASM

This occurs in the majority of instances in early adult life, depending somewhat on the nature of the growth, which are in the order given: tuberculoma, fibroma, sarcoma, glioma, carcinoma, and gumma, the first and last being really deposits of the specific granulomata. The tubercular tumour is more frequent in childhood, the gumma occurring between twenty and forty, and sarcoma at about thirty years of age, whereas carcinoma is apt to develop after forty years of age.

Brain tumours are in many cases distinctly hereditary, perhaps in 50 per cent of the cases. This fact has helped the writer in

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the diagnoses of obscure cases, which, put with the weight of other evidence, will always be of service.

Symptoms.—These are of two kinds: (1) general, and (2) special, local, or focal symptoms.

The *general* symptoms consist in mental excitement, vomiting, so-called reflex or cerebral vomiting, vertigo, choked disk and headache, the latter being of a dull character. The choked disk is present in between 90 to 95 per cent of cases, and is usually more intense in cerebellar lesion. It is much more frequent in neoplasm than in other organic affections within the cranium. The vomiting mentioned comes on without cause, being not accompanied by nausea, nor is it particularly related to the ingestion of food. It is designated *reflex* or *cerebral* or *central* vomiting.

Special local or focal symptoms are of importance in determining the location of the growth. If it be located in the frontal lobe, there will very likely be undue mental apathy, unless the tumour itself produces much irritation or protrudes into the orbital cavities, where, of course, ordinary methods of diagnosis will determine. The peculiar incoherency of mind in frontal-lobe disease is a symptom, if taken in conjunction with other signs of tumour, that is of great value. Keen has laid great stress upon tenderness over the site of the tumour. This is of value only in cortical growths, where it may be a distinct help.

If the tumour be localized in the **motor cortex** of the brain, there will be convulsions, focal in character, due to the location of the tumour, and later in its growth paralysis of the same parts will follow.

If the tumour be located in the **parietal region,** sensory changes may be found associated probably with mind-blindness or asteriognosis, depending upon the location of the growth.

A tumour in the **temporo-sphenoidal lobe** would produce word-deafness.

A tumour in **Broca's region** would produce motor aphasia.

A tumour located in the **corpus striatum** would produce a paresis of the opposite side without convulsions, and likely disturbances of temperature, due to the involvement of the centre for heat regulation within the striatum.

A tumour in the **optic thalamus** would produce hemianopsia, and within the anterior part the Wernicke pupillary inaction symptom; whereas, if posterior to the thalamus, the centre for pupil

contraction not being involved, we would not have the symptom present accompanying the lateral homonymous hemianopsia.

A tumour in the **corpus callosum** will produce symptoms similar to subcortical tumours from which they cannot be distinguished.

A tumour in the **cerebellum** produces a most marked choking of the disk, is typical, and in addition there is titubating gait, with other evidences of ataxia, frequently manifested in the hands, as athetosis. Knee-jerk would be vacillating and early increased. Later it may disappear, mostly in cases involving the middle lobe.

Gyrus Uncinatus.—Tumour here will produce perversion of sense of smell—anosmia, hyperosmia if involving the olfactory bulb or nerve.

Gyrus Fornicatus.—Tumour in this region will be evidenced by a loss of sense of smell.

Cuneus.—Tumour will produce, when located in this region, mind-blindness and hemianopsia without the Wernicke sign.

Sensation of taste may be disturbed when the tumour reaches the centre of the glosso-pharyngeal and facial nerves (chorda tympani).

Tumours of the meninges will cause a greater amount of pain than if the growths develop in the brain structure, since the sensory nerves are in the dura, and are therefore irritated.

Pathology of Brain Tumours.—This will entirely depend upon the nature of the growth, whatever that may be. There is always found about the neoplasm a subacute inflammatory process with round-cell infiltration and more or less serous exudate. The brain is apt to become œdematous from interference with the circulation. These points should be particularly remembered, since they give rise to symptoms which are necessary in the diagnosis of the case. In a case of Weir Mitchell's, the localization of the tumour was confirmed by autopsy, although the localizing symptoms were entirely masked in the later course of the affection by the circulatory disturbances already referred to.

Diagnosis.—This is a difficult matter indeed. The principal disease to be confused with it is abscess, where the history of septic process about the cranium will be an important point to be taken into consideration. The excursion of temperature above or below normal will also assist in the diagnosis. Chronic meningitis and sinusitis from the pain alone are likely to be confused

with tumour, but are wanting in localizing symptoms, and in the intensity of the choked disk seen in tumour.

Prognosis.—This is bad, but 7 per cent of the cases are operable, and just this per cent has a chance of recovery.

Treatment.—Beyond surgical there is little to be done; medical treatment is simply palliative, such as in the use of iodides, mercury, or other alteratives. Hygienic guarding of the patient is essential above that of all other brain affections. Pain must be controlled by morphia. A pill of camphor monobromate, gr. iij, often affords relief.

CHAPTER XII

INFLAMMATIONS OF THE MEMBRANES OF THE CORD AND UNCLASSIFIED DISEASES

External pachymeningitis involving the dura mater; internal meningitis involving the dura mater; leptomeningitis involving the pia mater; hypertrophic pachymeningitis involving both dura and pia.

Inflammations of the Dura (Pachymeningitis).—*a. Pachymeningitis externa* is a secondary inflammation, but is occasionally met in the acute form following caries, tumours, aneurysms, or syphilitic affections of bones. An abscess may penetrate the spinal cord, or the inflammation may be set up in the peridural tissue in long-standing cases of decubitus, pachymeningitis resulting.

Symptoms.—These are usually those of compression myelitis. The chronic form of external pachymeningitis is most commonly secondary to tuberculous disease of the spine (see Myelitis in Pott's Disease, Chapter X); hence the vertebral disease is important in the production of symptoms. This form is apt to be localized to the site of Pott's disease. The internal surface of the dura may be perfectly smooth and in some cases but slightly adherent to the arachnoid. The external surface is rough and covered with a cheesy substance. In some cases this material is chiefly anterior to, and in other cases it completely surrounds the cord.

b. Pachymeningitis interna is usually found located in the cervical region. The space between the cord and the dura is occupied by a form of concentrically arranged fibres, developing within the dura, and not on the outside at all. The condition is similar to hæmorrhagic pachymeningitis interna of the brain. The cord is usually compressed, the central canal dilated (hydromyelus), and there are secondary degenerations. The nerve-roots are in-

volved in the growths and are damaged and compressed. The disease is chronic and may be limited to one segment, or in the greater number of cases it actually involves a considerable portion of the cervical enlargement.

FIG. 53.—CHRONIC CERVICAL TUBERCULAR EXTERNAL PACHYMENINGITIS. (Specimen from case of complete diplegia in the Philadelphia Hospital.)

(a) Site of tubercular deposit at site of kyphosis; (b) cauda.

Symptoms. — Intense pains along the nerves whose roots are involved are present. There may be hyperæsthesia or anæsthesia with numbness and atrophy of the interossei, hypothenar and thenar groups. The arms become weak, the extensors may remain intact while the flexors are affected, producing the so-called " claw-hand " — *main en griffe*. At times the second stage also, of the French, consisting of involvement of the lower extremities and introduction of spastic paralysis, may supervene several months later, due to secondary degeneration in the cord. The disease runs a chronic course, lasting two or more years. In a few instances in which symptoms pointed definitely to this condition recovery took place, and I have no doubt that not a few cases of amyotrophic lateral sclerosis that have been reported much improved by several observers are cases of pachymeningitis interna with complete absorption of exudate.

Diagnosis.—(a) The external form is diagnosed-by the con-

currence of Pott's disease, the absence of atrophy of muscles, and the usual seat being in the dorso-lumbar region of the cord. (b) The internal type is diagnosed first from amyotrophic lateral sclerosis by the fact that severe pain does not occur in the latter; from syringomyelia by the absence of sensory changes characteristic of gliosis; and from tumours by the more localized symptoms in neoplasm.

Prognosis.—This is to be guarded, since some of the most marked cases have been greatly improved to the surprise of the neurologist. Given a case in which the disease has been stayed with physical health of the patient maintained, and where great contractures or atrophy of the part has not occurred, the prognosis may be guardedly given as favourable to partial restoration of the health, at least. Acute fulminating cases from the beginning usually end in the death of the patient. Even in cases that have apparently recovered any undue exertion or exposure to heat is apt to cause recrudescence. Treatment of either form is the same as for that of other types of meningeal inflammation.

LEPTOMENINGITIS

This is a condition closely allied to pachymeningitis physiologically, although pathologically pachymeningitis consists of inflammation of the pia. Practically these inflammations are associated more or less in every case. Leptomeningitis is more likely to be acute or fulminating, due to the fact that it is, as a rule, a septic type of inflammation, and also is resident in the larger blood-vessels of the pia associated more intimately with the nervous system. For this reason, even although the inflammation be not so severe, serious symptoms would result. The difference in the symptomatology between leptomeningitis and pachymeningitis would consist largely in the fulminating character of the former, accompanied by much less pain in the back or over the site of inflammation, for the reason that the sensory roots are less impinged upon; but there is greater paralysis in the parts below site of lesion, and also the greater liability to decubitus. Leptomeningitis is seldom a chronic disease, hence there are no special symptoms described for the latter, since that would be clinically the same in all respects as pachymeningitis chronica.

Prognosis.—This is uncertain. It will depend largely upon the

degree of infection and the extent of involvement of the cord.
Where there is early involvement of the multipolar nerve-cells
there is usually decubitus, accompanied by great palsy and disturb-
ance of the bladder as well as of the rectum, the patient usually
succumbing within a few days. Such cases rarely recover without
chronic myelitis and paraplegia being the result of the leptomen-
ingitis.

Fig 54.—Atrophy of Interossei, Thenar and Hypothenar Muscles of Both
Hands; (also of Shoulder Girdle and Pectoral Muscles) in a case of
Infectious Pachymeningitis Cervicalis Hypertrophica. (Medico-Chirur-
gical Hospital.)

Hypertrophic pachymeningitis involves both pia and dura.
It is usually septic in origin. The gonococcus may be the organ-
ism acting as exciting cause, as in the case under our care shown
in the illustration (Fig. 54).

Treatment of Pachymeningitis and Leptomeningitis.—This
consists of absolute rest in bed in a quiet, darkened room, the appli-
cation of ice to the spine, and the use of counter-irritants, such as

strong tincture of iodine over the site of the inflammation, of blisters, or the actual cautery, repeatedly applied, in chronic cases. In the acute form general antiphlogistic measures may be necessary, such as cold sponging or the cold pack; or by antipyretic drugs cautiously used, such as phenacetine and antipyrine. It may be necessary to give small doses of morphine to control the pain. If there are convulsions, the use of bromides and chloral, mixed, in fairly large doses, will be indicated. A 15-per-cent ointment of soluble metallic silver used by inunction in dram doses is a valuable remedy in septic cases. In chronic cases, if convulsions persist, administer ether for their control. If contractures follow, orthopædic treatment may be necessary, such as tenotomy or extension of the contracted limbs. This, of course, should be resorted to after it is evident that massage and Swedish movements prove of no avail. The use of potassium iodide is valuable as a remedy in chronic cases for the absorption of the exudate. This drug should be pushed to its physiological limit and continued for many weeks. In cases of paraplegia, resulting from pressure of exudate or actual inflammation through extension from the pia to the cord, the treatment will be that of the existent sequent myelitis.

CAISSON DISEASE

SYNONYM: *Diver's disease*

This is a disease with marked nervous manifestations, induced by the long submergence of the body in air at high pressure. It is doubtful if the affection would develop, until very late at least, were the body kept in a gradually increased atmospheric pressure.

Etiology.—As indicated above, the affection is caused by the sudden change from increased to decreased pressure of the atmosphere. It is probably due to the enfeebled vaso-motor nervous system. Divers are particularly subject to this, hence the name of diver's paralysis. The pressure that will induce diver's paralysis must be more than three atmospheres.

Symptoms.—It is characterized by a paraplegia, less frequently general palsy, appearing on returning from the compressed to the surface atmospheric pressure. These symptoms are more likely to develop on rapid change from the high to the ordinary atmospheric pressure. They may supervene shortly after leaving the

caisson or may be several hours in their development. In the mildest forms there are pains about the knees, which are often of great severity. Abdominal pain and vomiting are not uncommon. The muscles may be tender to the touch and the patient walk in a spastic fashion. Accompanying the other neurologic symptoms there is dizziness and intense headache, which later may, however, occur without vertigo. In the severer forms we may find complete motor and sensory paraplegia, a monoplegia or hemiplegia being rare. The most extreme attacks resemble apoplexy; profound coma, and death rapidly supervenes within a few hours.

Prognosis.—In cases of paraplegia the outlook is usually good. The palsy may pass off within a day or may continue for several months.

Pathology.—This is indefinite, although it is probably a vaso-motor disturbance in the simple cases that recover. In the severer cases there have been found, as in the case of Leyden, foci of hæmorrhages and evidence of acute myelitis. Fissuring of the cord sometimes occurs, some authorities believing it to be a constant phenomenon. It has been suggested that this condition is due to the freeing of nitrogen within the cord, the element having been absorbed by the blood under high pressure.

Treatment.—The patient should be kept quiet. Pain must be controlled by analgesics, such as morphine, etc. Inhalations of oxygen and the use of compressed air has been advised. Paralysis should be treated as any other form of palsy of similar type. As a prophylactic measure divers should pass gradually into the compressed-air chamber. According to A. H. Smith, at least five minutes should be allowed for each additional atmospheric pressure. Another prophylactic measure consists in bringing the patient very slowly into the normal atmosphere; as we have indicated should be the case in entering the compressed-air chamber. Ergot is a remedy which may control the vaso-motor nerves in cases where there is paralysis of vaso-constrictors. It should be given hypodermically in half-dram doses. Bandaging of the limbs has also been advised.

LANDRY'S DISEASE (ACUTE ASCENDING PARALYSIS)

This is a disease probably of toxic nature, more frequent in males, and coming on in cool weather in the greater number of cases, as though the temperature had something to do with its production. It is in all probability due to an autocthonous poison.

Symptoms.—A case of Landry's disease, or ascending palsy, if it be typical, consists of a fulminating onset of paralysis, beginning in the lower extremities, and ascending rapidly to the trunk and upper extremities, the head muscles escaping. It usually occurs between twenty and thirty years of age. The symptoms are frequently preceded by moderately high temperature. Nausea occurs, but gastro-intestinal disturbance is not a particularly notable feature of the disease. There is a sense of paræsthesia, to a slight degree only, in the extremities, but no other distinct sensory disturbances. While the reflexes are absent there is no tenderness of nerve-trunks and no involvement of the sphincters of the bladder or of the rectum. The patient is almost completely paralyzed, as above described, within seventy-two hours after the onset of the disease, and may not be able to move a muscle below the head; the chest expansion being very little, cardiac rhythm irregular, sometimes very rapid (tachycardia); the respiration being carried on entirely by the diaphragm through the phrenic nerves. The external respiratory nerve coming off lower down from the cord being affected, leaves the motor function of respiration alone to the phrenic nerve. Involvement of the pneumogastric nerve frequently occurs, which is another serious cause of embarrassed respiration, heart action, and cord function. This is often the cause of the sudden death which usually supervenes at the end of a week or ten days. There have been no cases recorded, such as those described, that recovered without leaving some disturbance or serious alteration of the nervous system as a sequelæ. A case under the writer's care may be referred to, where the man is to-day the subject of chronic muscular atrophy with *main en griffe* or claw-hand. So that cases that do recover are generally those where the symptoms will have been proved to be the result of some definite lesion, and not those in which the pathology has not as yet been understood or discovered. In some instances the paralysis may be *descending* in its initiation and sequent course.

Diagnosis is therefore as between this disease and multiple neuritis, if any difference at all exists, the only point being that the cases that have recovered present signs of multiple neuritis or involvement of the cord. The fatal cases, through the rapidity of their courses, do not develop pathologic changes that are demonstrable by modern microscopic technique.

Prognosis.—This should be put down as absolutely fatal.

Pathology.—In some cases diffuse myelitis or neuritis has been found. In others, the majority, no lesion has been detected. In one reported by the writer (Journal of Nervous and Mental Diseases, January, 1893), a multiple neuritis is the real cause of the clinical picture. Of course, these cases are taken out of the realm of Landry's palsy *per se*, but the clinical picture may be identical. A typical case of Landry's palsy consists of no definite lesion; nor does the patient develop trophic changes, such as bedsores.

Treatment.—This consists in supporting the patient by means of strychnine hypodermically, or by transfusion of blood by hypodermoclysis of normal salt solution, in order to counteract the condition of the blood, which is undoubtedly toxic. The use of galvanism to encourage respiration and cardiac tone would be of importance in the cases where these functions are particularly hampered. Rectal nourishment is a procedure which will be of value, since any attempt to give food *per orem* might excite dangerous symptoms of respiratory or cardiac failure. If the case should survive, the after-treatment would be as indicated in chronic neuritis or myelitis—i. e., massage, galvanism, alterative drugs, as iodide of potassium or the so-called Donovan's solution, the liquor arsenii et hydrargyri iodidi (a 1-per-cent solution), given in 3- to 5-minim doses thrice daily. We know of no better alterative remedy in chronic non-specific inflammatory states than this drug. The use of crutches may be necessary to get the patient about. In all chronic palsies we wish to again urge getting the patient into action soon, since use stimulates function; otherwise hysteria may soon become a part of the clinical picture.

CHAPTER XIII

DISEASES OF THE SPINAL CORD

MALFORMATIONS OF THE SPINAL CORD—SYSTEM DISEASES

(General considerations)

THERE are some thirty diseases of the spinal cord. The majority are organic and come under the head of inflammations and of degenerations or system diseases. Organic diseases the result of injury and inflammations are most common. Functional diseases of the cord alone are rare, such as the so-called "irritable spine," which latter, too, is associated with general functional diseases, as hysteria.

Etiology.—Most causes come under injury, auto-intoxications, poisons from without, exposure, infectious diseases, and excess of functionation. Middle-aged persons are more liable to these affections. Heredity may play a part in the predisposition due to vulnerable nerve-tissue.

Symptoms.—These all come under the head of irritation, depression, and of perversion. Irritative symptoms consist of pains, paræsthesias, hyperæsthesias, and feelings of constriction around the waist, rigidity, spasms, exaggerated reflexes, and irritability of the visceral and vascular functions. The symptoms of depression are those of anæsthesia, ataxia, palsies, wasting, and loss of control of viscera, as of the bladder. In cord disease paraplegia is the common form of palsy. Symptoms of irritability and depression often accompany each other. The more superficial, the more irritative signs exist; the more central, the more paralysis and visceral weakness prevail. In meningitis and meningeal affections there is much pain, and *vice versa*, as to cord diseases *per se*.

Pathology.—Inflammation of the cord membranes is not uncommon; but primary inflammation of the cord itself is unusual,

since most of the diseases that used to be called myelitis are secondary to injuries and softenings. Tabes and progressive muscular atrophy are given as "system diseases" by most writers, because they affect certain long-fibre tracts or groups of cells; but this name implies too many restrictions, hence it is retained only as a matter of convenience. Secondary degenerations alone are systemic. The cord is not infrequently the seat of abscesses, hæmorrhages, or tumours.

Diagnosis.—In making a diagnosis of cord diseases help comes through a thorough knowledge of cord functions. Physiology and anatomy point out the signs and symptoms more clearly than in any other part of the body. (See Chapter I.)

Prognosis.—Spinal-cord tissue once destroyed can never be renewed to more than a very limited extent. This regeneration applies to the nerve-fibres, not the cells. The diseased cord frequently adjusts itself to the pathological damage; but again, injury is very likely to extend by the process of secondary degeneration so rapidly that compensatory function cannot take place.

The **special diseases** of the cord are as follows:

(1) *Malformations*: Myocele, meningo-myelocele (spina bifida), meningocele, heteropia, amyelia, micromyelia, macromyelia, double cord.

(2) *Vascular disorders*: Anæmia, hyperæmia, hæmorrhage, endarteritis with aneurysm, embolism, thrombosis, œdema. *Secondary* to these conditions are *softenings, myelomalacia,* and *sclerosis.*

(3) *Inflammations*: Meningitis, myelitis, abscess. Also secondary softening and sclerosis.

(4) *Degenerations*: Primary: Locomotor ataxia, combined scleroses, hereditary scleroses, progressive muscular atrophy, and similar affections.

(5) *Syphilis*: Meningo-myelitis, gumma, single or multiple, and specific endarteritis.

(6) *Tuberculosis*: Miliary and solitary.

(7) *Tumours.*

(8) *Functional and toxic disorders.*

MALFORMATIONS

Spina Bifida (Rhachischisis Posterior)

This is a congenital hernia of the spinal membranes, sometimes of the cord, through a cleft in the vertebra caused by absence of the vertebral arches. It is therefore more of a malformation of the vertebral canal rather than of the cord, although this is not always so, for the cord and its membranes may be found in a condition of agenesis at the site of canal malformation.

Etiology.—About 1 in 1,200 children are thus affected (French statistics). It may be associated with hydrocephalus or with some other defect of development, such as imperforate anus or pharynx, or of ventral hernia. Heredity may be a factor in its causation. It is not due to dropsy of the cord, but is a true malformation. It is more common in males.

Varieties.—There are three forms described:

(1) Spinal meningocele is a condition in which the spinal membranes alone protrude into the sac.

(2) Spinal meningo-myelocele is a form in which the membranes and cord both pass into the sac.

(3) Syringo-myelocele (hydrorhachis interna) is a form in which the fluid is in the central spinal canal and the inner lining of the sac is formed by the meninges and thinned-out spinal cord.

Anatomy.—The most common are the first two types, which are called hydrorhachis externa. The fluid lies in the subarachnoid space, hence the wall of the protruding cyst is lined with the dura and arachnoid. The nerves and cord protrude into the sac in two thirds of the cases, forming a meningo-myelocele, but in some only a few nerves are found. When protruding into the sac they lie on the *posterior and median surface,* being attached and forming part of the wall; hence the spinal nerves start from the wall of the sac and go back into the vertebral canal. Besides the above-noted contents the tumour contains fat and connective tissue. The external surface is often red, flattened out, and smooth, and there is sometimes a depression on its median surface where the cord is attached.

Symptoms.—Spina bifida generally occurs in the lumbar and sacral regions, since the laminæ here are the last to solidify. But two or three vertebræ are usually involved. The tumour varies from 1 inch to 6 or 7 inches in diameter. It may be sessile or

be pedunculated. The outer skin is often glossy, or tough, thickened, or ulcerated.

Children with spina bifida are usually very poorly nourished or inherently feeble, and as a rule poorly developed mentally. In over 50 per cent of the cases paraplegia is found associated more or less with anæsthesia or involvement of the sphincter muscles. Contractions and contractures may occur, causing various phases of acquired talipes.

Prognosis is grave, being best for meningocele. The patient may live in such cases to early adult life, but injury or infection may occur, and the patient be carried off by secondary myelitis.

Diagnosis is fairly certain. Usually it is only necessary to exclude congenital tumours which happen to be located in the lumbo-sacral region. · The most important question to decide is whether the cord and nerves are present in the sac, which is very probable if there is much paralysis, anæsthesia, or sphincter trouble, or if there is a depression on the median external surface. The passing of an aseptic insulated needle connected with a galvanic battery may be tried in a difficult case.

Treatment is entirely surgical, and of avail only in meningocele. At present injections of Morton's fluid (iodine, gr. x; potas. iodid., gr. xxx; glycerin, $\mathfrak{z}$j), in dram doses, seem to be the most successful. These injections should be made on the outer portion of the sac, the child being kept on the back. Compression is dangerous if the fluid has been previously withdrawn. Opening the sac, excising, or ligaturing are most dangerous, especially if part of the cord should happen to be in the sac. In skilled hands surgical results have recently been more favourable. Operation should not be done within the first four months of life.

MENINGOCELE OF THE CORD

Meningocele of the cord is protrusion of some or a portion of the membranes of the cord from the canal. It usually occurs in the infant, being a congenital defect of closure of the neural canal, the bones and membranes themselves being involved. In some cases the cord itself protrudes.

Symptoms.—No symptoms exist except the physical signs of protrusion of the tumour (which is usually in the lower lumbar, or in the sacral region of the cord) until at some later period in

life injury to this mass sets up an inflammation, when the symptoms of spinal meningitis and paraplegia will follow. The worst cases, due to the defect of the nervous system as well, will present typical signs and symptoms of paraplegia, with contractures, loss of sensation, involvement of the sphincters, bedsores, etc.

Prognosis.—This will depend upon the degree of defect and the condition of the patient. More cases may pass through life undisturbed, but the existence of a tumour about the sacral re-

Fig. 55.—Agenesis and Micromyelia of Spinal Cord in a Male aged Forty Years; also Lack of Development of the Acoustic Nervous Centres. (Philadelphia Hospital.)

gion is always a menace to life. Cases with paraplegia at birth will be very apt to succumb within a few months to a year.

Treatment.—As in cases of spinal meningocele, treatment would be mainly surgical, if at all. This consists in compression and protection of the part. Removing the protruding mass, or the injection of carbolic acid or some other irritant into the tumour mass, with the hope of causing its disappearance, should of course only be done in selected cases.

Heteropia is a rare malformation wherein masses of gray matter are found in abnormal positions. Van Gieson describes a false heteropia that may be caused by manipulation of the cord after death, in which case the displaced masses consist of nerve-cells or neuroglia.

Amyelia, or absence of the spinal cord, can exist only when
    15

the brain is absent, although absence of the brain may occur without absence of the cord. In amyelia the spinal nerves are usually present. Amyelic monsters cannot live.

Double cord is very rare, and involves only a part of the cord except in cases where there is a double vertebral canal.

Double central canal is not rare. It usually is found in only a part of the cord, and the two canals lie parallel.

Asymmetry of the cord, usually due to abnormality in the course of the pyramidal tracts, is not so rare.

Splitting of the cord and defects in development at special levels are found occasionally.

Agenesis implies lack of proper development of the cord. (Fig. 55.)

Micromyelia is a condition in which the cord is abnormally short or is small in size. The normal cord varies in diameter from 6 to 9 millimetres (dorsal), 8 to 11 millimetres (upper cervical). 15 millimetres (cervical), and 12 millimetres (lumbar).

SYSTEM DISEASES

By reference to the chapter on Anatomy will be found the division of the spinal cord into certain tracts or systems. A degenerative disease of these tracts is frequently confined to one or may extend to two tracts. The first is designated a simple system disease, and the second is styled combined system disease. Both the peripheral and central motor neurons may be involved, singly or together.

LOCOMOTOR ATAXIA

(Tabes dorsalis; posterior spinal sclerosis)

Locomotor ataxia is a system disease, affecting the afferent or sensory tracts of the cord.

It is divided into the following stages:

1. Preataxic.
2. Ataxic.
3. Paralytic.

The prominent symptoms, as its name indicates, are ataxia, inco-ordination of gait, sensory disturbances, loss of reflexes; also accompanied by atrophic disturbances and impairment of the special senses. In tabes the bones are brittle.

The symptoms confined to the *preataxic stage* are of shoot-

ing pains, coming on at irregular intervals, and usually in the legs, and principally in the calves. The patient may complain of a sense of numbness in the extremities, and may or may not have a sensation of constriction about the body, which is called the girdle-sense, or, if painful, it is called the " girdle-pain." The patient even now may develop the perversion of sensation, as of something soft, when really walking on a hard surface like a pavement. At times there may be violent paroxysms of pain referred to certain internal viscera, the so-called " gastric crisis " being the most important and frequent one. This pain is located in the epi-gastric region, and is accompanied by vomiting and excessive secretion of the gastric juice. The attack may last from a few moments to an hour or so. In severe attacks the prolonged weakness following may exist from one to several days. The sexual power is generally decreased in this stage, although the appetite is frequently increased. There may be difficulty in urination. Upon examination of the eye, the so-called Argyll-Robertson pupil may be determined —this consisting of a lack of response of the iris to light stimulus, although accommodation reaction is preserved. In some rare cases there may even begin optic atrophy in this stage of the disease.

Ataxic Stage.—This is the stage when the patient usually is seen by the physician, from the fact that he does not report for treatment until inco-ordination of gait actually sets in; or frequently from lack of diagnostic acumen of the physician the disorder is not made out until this stage is reached. It is characterized by the ataxic gait, which consists in throwing the limbs forward, outward, and downward in an irregular and lax fashion, the heel descending first. The gait is more ataxic when the patient's eyes are closed or when he is walking in the dark. Since the cause of this symptom is the perversion or diminution of the muscular sense, the explanation of a better locomotion in the light or with the eyes open lies in the fact that the eyesight makes up for the impaired muscular sense. Another symptom present and dependent upon the same disorder of co-ordination is the Romberg's sign, which consists of marked swaying of the individual, the feet being in the position of attention. The sway is more pronounced so soon as the eyes are closed. Other evidences of inco-ordination are shown in muscular movements generally, and particularly in these finer movements, as of the fingers, the patient being unable to write properly, the letters being slowly and irregularly outlined.

If the index-fingers of opposite hands are brought together there will be found to be great inco-ordination as they approach each other, or if brought to the nose the same phenomenon will be observed; this sign being also exaggerated by the patient clos-

FIG. 56.—STANDING POSTURE, STATIC ATAXIA IN TABES DORSALIS. (Philadelphia Hospital.)

ing his eyes. A variety of ataxia called *static ataxia,* frequently existing, is detected by having the patient lie on his back, then elevating the legs from the bed in a fixed position. If inco-ordination exists the patient is said to have static ataxia. Or it is tested for by the patient standing erect, eyes closed. If he sways badly, he also has static ataxia. The latter is the Romberg sign, as indicated above.

In this stage there generally is an aggression of all the other symptoms mentioned, though in the minority of cases the painful sensations become abated, and paræsthesia will become lessened or disappear in them. The patient loses his vigour, and there is usually muscular wasting and weakness, in spite of preservation of the digestive function. It will be found in 50 per cent of the cases that the optic nerves degenerate, this being a *primary optic atrophy* and not accompanied by the choked disk of optic neuritis. The ataxic stage may last for many years, and during that time periods of exacerbation and remission of the symptoms occur, out of proportion apparently to the extent of lesion. This irregularity in such cases must be due to loss of nutrition for the time being, rather than to organic change in the cord.

Paralytic Stage.—This comes on very late in the course of the disease, and signifies muscular paralysis, associated with signs of paresis. In this stage the patient becomes bedridden; develops more or less atypical paresis or progressive paralysis of the insane. A very small per cent of cases reach this stage, since they are usually carried off by some intercurrent malady. The absence of the deep reflexes determined so early in the preataxic stage remain persistently absent during the entire course of the disease. In some cases optic atrophy is an early symptom, and is so rapid in its onset and progress that this class of cases is given a distinctive name, the so-called *amblyopic* form of tabes. In these cases it is frequent to find the ataxia held in some subjection or actually bettered with the onset of blindness. This is marked in a coloured man in my wards at the Philadelphia Hospital, where on becoming totally blind the ataxia is much lessened; so with the crises from which he suffered up to that time. Other cases of tabes may develop unusual symptoms, such as perforating ulcer of the foot, which is a very rare condition, occurring in not more than two per cent of the cases. There is a typical case of this complication in ataxia, a man, aged forty, now in the paralytic stage of the disease, at the Philadelphia Hospital, a description of which ulcer will be typical of the condition. The ulcer extends from the base of the great toe on the sole of the foot, over the ball, is much indurated, but signs of inflammation are very slight; the nutrition of the surrounding tissue being at a low stage. The ulcer is sharply cut, $\frac{1}{2}$ by $\frac{3}{4}$ of an inch in diameter, and extends down to the depth of $\frac{5}{8}$ of an inch by a central sinus which leads down to the metatarsal bone. There is also a foul odour emanating from the sore, with considerable sanious discharge therefrom. It is not at all painful.

Another unusual development in tabes is the *arthropathy*, which consists in enlargement of the joint, generally a large one, as the knee, due to a subacute trophic inflammation. The bones become porous, the synovial membrane lustreless or entirely disappears, the ligaments of the joints becoming so relaxed as to permit of easy dislocation. Synovial fluid occurs in excess early, but may later be absorbed, leaving a much relaxed joint. These joints are not painful, and only give disturbances by the enlargement and weakness entailed. The hypotonia of the muscles about the affected joint exaggerates the deformity. (Fig. 57.)

FIG. 57.—CHARCOT JOINTS (RIGHT KNEE AND BOTH ANKLES) IN TABES. (Medico-Chirurgical Hospital.)

The characteristic feature of all cases of tabes is the disproportionate increase of inco-ordination to the small amount of muscular weakness *per se* and wasting; so that in some cases the patient will show very good strength of muscle when the ataxia is pronounced. Absence of the Argyll-Robertson pupil, the other symptoms being present usually, but rarely occurs.

Other unusual symptoms are the areas of anæsthesia, which are generally irregular in outline, and are situated upon the chest, over the shoulder girdle, or upon the upper arm, and disobey the anatomical rules in that they do not follow the nerve-trunks, nor apparently the segments of the cord in all cases.

The *duration* of tabes will be on an average from three to thirty years. The disease is much more frequent in males, occurring at about thirty years in an average case.

Etiology.—It is a parasyphilitic infection in over 90 per cent of cases, usually occurring from eight to fifteen years after the initial lesion; other *causes* being overwork, auto-intoxications, or chronic poisonings, such as of malaria, of which I have seen one case in a young man eighteen years of age.

Pathology.—The pathology of tabes consists in sclerosis of the posterior columns of the spinal cord and the nerve-roots. The columns of Clarke and Burdach are particularly involved. The spinal ganglia and roots are frequently affected. The peripheral nerves are occasionally diseased. The pia mater between the posterior roots is thickened and opaque, the posterior roots being enlarged, while in advanced cases they become thinner and more translucent. The spinal cord is reduced in size, and the posterior columns appear to be shrunken and present a grayish appearance. The first sclerotic areas, as a rule, are found in the posterior roots, usually in the lumbar region and in the tract of Lissauer; next the columns of Burdach are affected, beginning along the median side of the posterior horns, spreading out towards the posterior median sæptum, finally involving, as inferred, the column of Goll; and may ascend to the upper thoracic and cervical regions, the usual seat of lesion being confined to the lumbar cord. The fine fibers running from the posterior roots to the column of Clarke are also sclerosed, but the cells then are not usually destroyed, and consequently the direct cerebellar tract is rarely degenerated.

Diagnosis.—Tabes should not be confused with any other dis-

ease, save possibly syringomyelia, where the dissociation of sensa-
tion in the latter and muscular atrophies would be distinguishing
points.   Chronic multiple neuritis, with ataxia, would be deter-
mined by the muscular atrophy again; also by tenderness over the
inflamed nerves, so that in studying a given case the presence of
shooting pains, absence of knee-jerks, the presence of Argyll-Rob-
ertson pupil with or without optic atrophy, and history of syphilis,
there would be very little doubt as to tabes being present.   There
is increase of knee-jerks in ataxic paraplegia.

Prognosis.—While complete recovery rarely occurs, much can
be done, however, to ameliorate the symptoms and to lengthen the
period before the paralytic stage should occur.   Ten years is an
average duration.

Treatment.—In an early case usually antisyphilitic measures
are indicated, since it is possible that some remnants of the patho-
logical exudate of the third stage of syphilis should exist, which
the use of the iodides and mercury by the sorbefacient and altera-
tive action may tend to dissipate.   Prolonged active antisyphilitic
treatment should not be carried on after it is noted that distinct
improvement has not occurred.   In debilitated cases a combination
of cod-liver oil with the alterative drugs, as arsenic, should be em-
ployed.   In anæmic persons the use of iron is indicated.   If after
a thorough trial benefit is not had, some of the following drugs
may be used until the one is determined that particularly benefits
the case.   Arsenic given in the form of Donovan's solution, 4 drops
t. i. d.; or the chloride of gold and sodium, $\frac{1}{48}$ gr. t. i. d.; and the
glycero-phosphate of lime or soda.   Long-continued doses of sil-
ver nitrate, $\frac{1}{4}$ gr. t. i. d., have been found efficacious by some ob-
servers.   Strychnine may be resorted to in cases where there is
much weakness, although, as a rule, strychnine is not an ideal
drug, often making the patient irritable and exaggerating his
ataxia on account of muscle stimulation.   In addition to drugs
the patient should be instructed to abstain from alcohol, tobacco,
and sexual indulgence, and he should have plenty of nutritious
proteid food, fresh air, and sunshine.   The overuse of the lower ex-
tremities, as in a half-hour's walk, is baneful.   Exercises should be
rather of a passive nature; hence the ideal treatment for a case of
tabes is the "rest cure," plus the use of massage and Swedish
movements by an experienced manipulator.   In mild cases, where
the patient is going about, the method first introduced by Weir

Mitchell and later amplified by Fränkel is important. This consists in a series of educational movements, the patient lying on his back and approximating his toes and his fingers together, etc.; finally walking along a crack in the floor and stepping over bricks placed upon the floor, with the eyes opened, then closed. As the patient improves he is instructed to do more difficult exercises, such as walking backward under the same conditions.

Warm sponge baths given at night are valuable in the relief of pain, and by the stimulation of secretion to the carrying off toxines from the system. The warm bath (temperature of 150° F.) must not last longer than fifteen minutes, and should be followed by a cold spray down the spine for two or three minutes, the temperature of the water being about 60° F. The entire procedure thus far should be followed by a brisk rub with a Turkish towel, in order to stimulate the superficial circulation, and thereby to relieve congestion about the posterior roots of the spinal cord. Another measure not used sufficiently for the relief of tabes, and especially the painful cases, is to resort to high altitudes, above 5,000 feet, in a dry climate. Such cases I have seen benefited in Colorado and New Mexico.

Suspension treatment is a valuable measure also in some cases to relieve pain. It consists in suspension of the patient by lifting the body to the tip-toes by means of a head-piece about the chin and occiput and under the elbows. The *séance* should last three to five minutes once or twice a week. It relieves pain by stretching the spinal ligaments and aiding circulation about the nerve-roots.

Drugs to relieve pain should be only used with great precaution for fear that the patient may form the drug habit. Antipyrine can be given in 5-grain doses; a combination of the analgesics with codeine, $\frac{1}{8}$ to $\frac{1}{4}$ grain, is frequently very valuable; a hypodermic injection of morphine, $\frac{1}{4}$ to $\frac{1}{2}$ grain, guarded by atropine, $\frac{1}{360}$ of a grain, must be given to control crises. To relieve the tingling and numbness of the legs static electricity administered thrice weekly is good treatment; but more particularly is the use of the Faradic " dry brush " an excellent procedure in these cases. Counter-irritation by blister or cautery over the lumbar spine at times acts remarkably well in alleviation of painful paroxysms.

Of great importance is the nutritive state of the patient, which should be carefully watched at all times even to the holding

in abeyance all other treatment, since if nutrition is not preserved improvement will not take place. Red meats, eggs, oysters, cereals, good bread and butter and milk, in as large quantities as the patient is able to digest, are the ideal foodstuffs that should be employed.

DISEASES OF THE MOTOR NEURONS OF THE SPINAL CORD

Primary Lateral Sclerosis

This is a disease characterized by a paralysis of the muscles, beginning usually in the lower extremities, and attended with increased reflexes, but unattended by muscular atrophy or sensory disturbances. This disease occurs most frequently between the ages of twenty and forty. It may appear earlier, as reported in a case by H. N. Moyer in a child of five years. I have seen for some years a case with Sinkler, reported by that gentleman and myself in a paper on family diseases, already referred to. In this case—a woman about thirty-eight years of age—the disease began about twenty years ago, and has been very slow in progress. Some cases have been supposed to follow syphilitic infection, or to be caused again by hereditary syphilis or alcoholism transmitted from the parents. Others have appeared to be due to traumatism to the back, or to exposure to cold and wet, and to certain infectious diseases; or to excesses, predisposing to general physical debility through excessive drain on the nervous system.

Symptoms.—The patient first complains of rigidity with weakness of the muscles of the lower extremities, which may be associated with a sense of fatigue or dull pains, but never of an active character. The symptoms may first commence in one leg before the other is at all affected, or one may be affected more than the other. The loss of power is very gradual and not at all in proportion (as in ataxia) to the impairment of progression, which in this instance is due to spasticity. The extremity soon becomes rigid, so that on flexing the joint a sensation as of bending a piece of lead pipe is transmitted to the examiner. If the movement is continued the rigidity lessens. The gait in the very beginning is slightly spastic, as shown by the patient wearing the shoes out at the toes or complaining of tripping over slight impedi-

ments.   Later the characteristic spastic gait prevails, and the feet appear glued to the ground and are pushed forward with effort.   The toes are raised over obstacles with great difficulty. Voluntary effort increases the clonic and tonic spasms of the muscles, and owing to spasm of the adductor and stronger muscles the knees are kept close together, and in aggravated cases there is often cross-legged progression.   The knee-jerks are markedly increased and ankle clonus well developed, although not so large as in cases of insular (or disseminated) sclerosis.   The superficial reflexes are slightly increased.   Muscles are not wasted, excepting that resulting from disuse.   They feel firm to the touch.   The sphincters are not involved until very late in the course of the disease.   Sensory disturbances are absent, other than paræsthesia, the result of circulatory disturbances from vaso-motor weakness.   The upper extremities do not become involved until late in the course of the disease.   Seldom do trophic ulcerations occur, but the affected extremities are very easily frost-bitten.   The patient may live many years in comparative comfort, other than the physical distress of being thus disabled from active life.

Pathology.—This consists in a degeneration in the pyramidal tracts.   That this may occur primarily is not definitely settled, and many observers believe that degeneration of the pyramidal tracts alone cannot occur, except as a secondary degeneration due to lesion higher up.   A few cases have been reported, however, in which no such primary lesion could be found.

Diagnosis.—In transverse myelitis sensory symptoms are more pronounced, the disease is more acute, anæsthesia frequent, and the sphincters are early affected.   In ataxic paraplegia the spasticity of the muscles is not so marked and evidence of lack of co-ordination is present; involvement of the sphincter is also common.   In amyotrophic lateral sclerosis there is muscular atrophy, not noted in the disease under consideration.   Secondary degeneration following cerebral apoplexy may rarely be mistaken for cases of primary lateral sclerosis, in which one side is more affected than the other.   The history of a previous apoplexy would, of course, settle the question.   Hysterical paraplegia may quite often be difficult to distinguish.   In lateral sclerosis rigidity of limbs is more pronounced when they are extended, and *vice versa,* a condition that does not occur in hysteria.   Also the deep reflexes, while they may be increased, are not so much so in hysteria.   True ankle clonus

is exceedingly rare in the disease under consideration. Other evidences will also be found, such as "stigmata" or hysterical paroxysms, which are more frequent, too, in females.

Prognosis.—The prognosis of the disease is steadily slowly onward and downward until the power of progression is lost. The patient may live for twenty or thirty years, and die as the result of an intercurrent disease, although involvement of the bulb may through inhibition of the vagus cause death from heart failure.

Treatment.—This is not very satisfactory. Nitrate of silver, chloride of gold and sodium, iodide of potassium, etc., may be of service. Massage is a valuable agent in arresting muscular wasting. The electric brush is also of use in stimulating the circulation. The patient should be well fed, given the hypophosphites from time to time, and must be well protected with woollen clothing. Strychnine is contra-indicated, as it excites the muscular spasms in already irritable muscle fibres.

SPASTIC SPINAL PARALYSIS

Spastic Paralysis

This term applies to a form of paraplegia caused by (*a*) chronic myelitis, as well as to a (*b*) congenital disorder in which there is sclerosis of the lateral columns of the cord. There has been much controversy as to the latter, some giving the name of Little's disease to the diplegic or paraplegic forms of the cerebral palsies of childhood, though, properly speaking, this should be applied to lateral sclerosis. Little first described the condition in 1846 and in 1877. In 1873 and in 1879 Dr. E. C. Seguin, of New York, described a condition of similar type which he called " tetanoid paraplegia." Erb and Charcot in France independently in 1875 published articles on " spasmodic spinal paralysis " and " spasmodic dorsal tabes " respectively. After this there was much confusion. Finally, the spastic paralysis has been shown to be due to spinal, associated probably with cerebral defect.

Etiology.—It is congenital and due to lack of development of the pyramidal tracts, which leads to a sclerosis of the lateral columns and to symptoms of spasticity of legs and arms, exaggerated reflexes, with some weakness, and at times slight atrophy. Prenatal or natal in its origin, it is primarily an agenesis in the first

instance; or if caused at birth, as Little supposed, may be due to premature or forced deliveries. It may be a family disease.

Symptoms appear shortly after birth, usually within a year, but may appear after the fifth year in family types, or even after maturity. There is a type of this disease I have seen where the spasticity remains entirely in the lower extremities, coming on at about fifteen years of age, and not affecting the upper extremities at all. In the ordinary type it is, as inferred, difficult to distinguish spastic spinal paralysis from the cerebral palsies (or " birth palsies," so called). The lack of marked mental impairment is a point in differentiation, the brain seeming to be spared except for its motor areas being damaged. At times, too, lateral sclerosis cases occasionally gain in development, and some increase in the use of the limbs follows more than in cerebral palsies. The so-called " scissor-legs " frequently are found in these patients, the crossing of the limbs being exaggerated in the efforts of walking. The arms are much less affected than the legs. The throat and facial muscles may be slightly involved. In some cases the disability increases quite rapidly. There is no pain, however. The arms may later be contractured. Finally the patient becomes helpless. Epilepsy or mental deterioration may develop at the time of puberty or adolescence.

Prognosis.—The mild cases that learn how to walk should be kept carefully at the practice of their muscles, when they may slowly improve and reach average health and adult age. Severe cases rarely reach adolescence, and generally die of some intercurrent disease before the twenty-first year of age.

Diagnosis.—It is distinguished from the cerebral (or birth) palsies by the absence of any material initial mental defect, microcephalus, or of epilepsy. From compression myelitis spastic paralysis is distinguished by the affection of the arms, the absence of pain, and of disturbance of the sphincters. Hereditary spastic palsy, which is also found to occur in families, begins later (at about the fifth year, see p. 238), and involves the legs chiefly.

Treatment consists in orthopædics, massage, and Swedish movements to prevent contractures and deformities. General nutrition must be carefully maintained; also the protection of the body from cold or extremes of temperature. Employment of tenotomy may be necessitated where mechanical treatment fails, in which case

braces should be applied to retain the normal position of the limbs, and the child then encouraged to walk about by means of the wheel crutch or other form of apparatus. Many cases have been improved so as to walk with some facility by persistence in this regard, and a hopeless life of invalidism stayed.

HEREDITARY SPASTIC SPINAL PARALYSIS[1]

This disease is of the family type, affecting different members of succeeding generations. It begins about the age of five, affects mainly the legs, runs a very slow course, is not accompanied by pain, ataxia, or visceral symptoms, and may continue for thirty years or more. The writer has two cases of this rare affection under observation where there seems to be a remission of all symptoms within the last six months.

FRIEDREICH'S DISEASE, OR HEREDITARY ATAXIA

This is a developmental disease, occurring in early youth, is more frequently met with in males than in females, and the symptoms very frequently begin at about the tenth year, the patient having been in apparent good health up to that time, although undoubtedly of vulnerable nerve protoplasm. There may be found a history of heredity, although the affection is never congenital; or there will be a history of syphilis or alcoholism in the progenitors, usually in the parents.

Symptoms.—These consist first in ataxia, very much like that of tabes, but accompanied by irregular choreiform movements, perhaps athetosis, as in a case reported by Swan and myself (Philadelphia Medical Journal, January, 1896). The speech is frequently affected in this disease, staccato-like and drawling, accompanied by dribbling of saliva from the mouth, inability to masticate food properly, and by dysphagia. The mental condition remains fairly good, although one is apt, from the mechanical defects, to appear feeble-minded, a condition similar to the pseudo-imbecility in some cases of spastic paralysis of childhood, where mental failure is exaggerated by the fact that the vocal muscles are

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[1] In all these so-called spinal spastic paralyses occurring in early life there must be some cerebral defect or agenesis, though the degeneration is largely associated with the lateral tracts of the spinal cord.

spastic. The Argyll-Robertson pupil is not so constant as in tabes dorsalis, but there is often present nystagmus, usually bilateral. The knee-jerks are not constantly absent, though usually so, and may change from year to year in accordance with the involvement of the posterior columns, lateral columns, or anterior horns of the cord in varying degrees, and thus disturbing the physiological reaction of these parts as the disease picture changes. The patient may also develop club-feet—the equino-varus position being particularly common—due to contraction of the posterior leg muscles. Localized muscle atrophy is in evidence, and where this occurs fibrillary twitchings will also be found. Curvature of the spine is another symptom that may exist, dependent upon muscular weakness of the erector spinæ group. Trophic joints are very rare indeed. Paræsthesia may exist. In Marie's cerebral type cerebellar gait exists.

Pathology.—This consists in an overgrowth of neuroglia cells within the central nervous system, particularly involving the posterior roots and posterior columns of the spinal cord. In addition round-cell infiltration may be present in other parts of the cord about the anterior horns and in areas diffused throughout the brain. It is undoubtedly the remnant of an embryological defect in the child, which by the developmental period first gives expression to symptoms, just as syringomyelia gives rise to symptoms due to a gliosis commencing in prenatal life from defect in the embryo itself.

Diagnosis.—This disease should not be confounded with any other. Diagnosis may lie between it and spastic paralysis of childhood, which latter presenting increase of reflexes in the spastic condition with more marked athetosis and lack of atrophies, would be sufficient signs for the proper diagnosis of this disease. Insular sclerosis is told by the more pronounced nystagmus, increase of reflexes and its later development. From tabes, the earlier development of Friedreich's disease, the lack of crises, and the type of movements (choreic) will make it clear.

Prognosis.—The patient may live for many years, but usually does not reach maturity.

Treatment.—This is solely palliative, and consists in proper nutrition; hygiene, as to clothing and exercise, which latter should be limited, though systematic, and particularly in the form of

calisthenics towards preventing contractions; for which massage and Swedish movements are also valuable agents. Drugs, such as potassium iodide, in small continuous dosage, also the chloride of gold and soda, $\frac{1}{48}$ gr. t. i. d., and in atonic cases the use of strychnine will be of great service. For the prevention of deformities, or where they exist already, the use of a " brace " may be necessary to the spinal column or to the lower extremities, and in some cases the use of crutches will be of value in permitting the patient to move about. These subjects are very liable to become bedridden, so that educational movements should be faithfully tried.

CEREBELLAR ATAXIA

This is also an hereditary disease, and begins later in life than Friedreich's disease—from fifteen to eighteen years of age.

Symptoms.—It is particularly characterized by the cerebellar or titubating gait added to the other symptoms of ataxia. The knee-jerks are increased, diminished, or precocious in this disease, but there is seldom found ankle clonus. The duration of the affection may be for many years (eight or ten), the patient generally dying of some intercurrent affection. Club-feet are very apt to develop, due to contractions, the gait being somewhat similar to that of ataxic paraplegia. The patient presents no sensory symptoms.

Pathology.—This consists in degeneration of cells of Purkinje in the cerebellum and the posterior columns of the spinal cord. Atrophy of the optic nerve may exist and nystagmus may be present.

Treatment.—This is symptomatic. The use of the hypophosphites and nutritional measures with calisthenics and Swedish movements as described under the previous disease. For the prevention of deformities of the legs braces should be applied by the orthopædic surgeon.

FUNCTIONAL DISORDERS OF THE SPINAL CORD

Functional disorders of the spinal cord include those of irritation or of depression coming under the head of spinal neurasthenia, or spinal irritation, or spinal exhaustion. These symptoms are described under the head of Neurasthenia (see Chapter XVI).

CAUDA EQUINA

The cauda equina is made up of five lumbar, from five sacral and one coccygeal (Müller), nerve-roots. They lie within the dura, extending beyond the spinal cord some 15 centimetres. These distinct motor and sensory roots do not quite unite until they pass out of the dura. The cauda begins at the lower edge of the second lumbar vertebra. The term *conus* is applied to the part of the cord below the second sacral segment. In this part (conus) the anterior root-fibres are smaller and fewer in number than the posterior or sensory root-fibres. Müller gives the arrangement of the visceral centres as follows: Second sacral, erection centre; third sacral, ejaculation centre; fourth sacral, bladder (detrusor) centre; fifth sacral, sphincter ani centre.

Symptoms and Diagnosis.—The diagnosis of cauda lesions involves a study of:

Fig. 58.—Tumour (Psammoma) springing from Dura, Lower Dorsal Cord. (Specimen from Philadelphia Hospital.)

1. Lesions of the lower end of the cord.

2. Cauda compression, irritation, or destruction.

3. Lesions of the peripheral nerves.

1. *Lesions of the lower end of the cord* generally come on rapidly, in a few days, following myelitis, etc. There is little pain, and dissociation of sensation comes on late.

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Fibrillary contractions and involuntary twitchings of the leg muscles occur. * Paralysis rapidly appears, involving the lower limbs in accordance with the segmental distribution of the nerves. It is a flaccid palsy and is followed by atrophy. The visceral centres are involved. If the conus is not implicated the paralysis does not seriously involve these centres nor the muscles of the pelvic girdle. The *motor symptoms are far more prominent* than sensory symptoms. The cord being small is easily destroyed, and very little improvement ever occurs.

2. In *disease of the cauda,* since it is usually a neoplasm, the symptoms generally come on slowly. In injury the symptoms appear rapidly after the trauma. There is often severe pain in the bladder, in the sciatic distribution, and it is bilateral. Anæsthesia in the area of the sciatic nerves follows later. The paralysis is slow in development. The *sensory symptoms are far more prominent* than the motor at all times. The sexual bladder and rectal centres are later paralyzed. Sometimes prompt operation, as in tumour, will give quick relief. This should always be done where tumour is suspected, otherwise the case is hopeless as to cure. The symptoms in compression of the cauda without destruction are like compression of the cord, but there is less motor disturbance, and there may be no involvement of the sphincters.

3. *Lesions of the peripheral nerves* (neuritis or injury) is rapid in onset. There are sciatic pains—tender spots along the nerve-trunks. The lesion may be only *unilateral,* the pain being not so severe, and there is no marked anæsthesia. There is little or no paralysis of the visceral centres, the sensory slightly predominating. There is often a history of sciatica or *alcoholism* or injury. Examination may disclose the presence of a tumour or of some disease affecting directly the sciatic plexus; and the prognosis is favourable.

DIFFUSE AND FOCAL DISEASES OF THE SPINAL CORD

SPINAL HYPERÆMIA, ACUTE AND CHRONIC

Ætiology.—Violent physical exertion, sexual excesses, amenor-rhœa, and certain poisons, as strychnine, are causes of spinal hyperæmia. It is also the first stage of acute inflammatory diseases. *Chronic spinal hyperæmia* is rare. It may occur in the membranes as the result of meningitis or injury. There is no absolute knowledge of separate chronic hyperæmia of the cord.

Symptoms are of a sense of weight around the loins, of heaviness, twitching of the muscles, a numbness, formications, and actual pain of a neuralgic nature, accompanied by numbness of the lower extremities. The sphincters may be involved. The symptoms are as inferred, usually confined to the lower extremities. The position of the patient, as mentioned by some authors, can have but little influence upon symptoms due to spinal hyperæmia. The signs and symptoms of chronic spinal hyperæmia are much like those of spinal irritation. They will be described under that head.

Pathology.—The circulation of the blood in the spinal cord, as has been shown in the chapter on Anatomy, is one which is difficult to disturb; but if so, it is slow in being brought back to normal. The violent activity of the heart and great increase in arterial pressure, and then the weakened state of the heart muscle and lowered tension, appear to modify but little the spinal functions. Hence it is unlikely that the large number of clinical symptoms that have been attributed to the circulatory failure are so caused.

Treatment.—This consists in the application of cups to the back, rest in the horizontal position, better upon the side or face, counter-irritation to the spine, or the use of cold, as in the form of ice-bags. Among drugs the employment of bromides and morphine is good treatment. The sorbefacient and alterative effect of·

chloride of ammonium and the iodides is recommended; or the use of galvanic electricity, the negative pole being placed over the spine and the positive at some indifferent point (5 minutes).

SPINAL ANÆMIA

Spinal anæmia is less known in its cause, symptomatology, and course than is hyperæmia. It is more apt to affect the posterior columns than the ventral aspect of the cord, for the reason that the dorsal nutritive arteries are smaller than the anterior ones. Severe hæmorrhages or extreme diarrhœas may be predisposing factors by causing general anæmia. Or aortic obstruction, as in aneurysm, may be the cause (through cutting off blood supply to the cord) of almost total abolition of cord function. It is not common for serious disturbance of the cord to persist from anæmia excepting where there is disease of the arteries, and in that event the sclerosis of the posterior columns may follow in the wake of an added pernicious anæmia, in which latter the general symptoms overshadow those of the spinal cord. The test of the patient's improvement when lying on his back is perhaps incorrect. Some writers associate with spinal anæmia a class of symptoms such as pain in the back and weakness of the legs,-which may amount to paraplegia, a group of symptoms that has been given the name of *spinal concussion*, but it is impossible to scientifically state whether spinal anæmia is the underlying condition in such cases.

SPINAL HÆMORRHAGE

Spinal Apoplexy

This general name may be given to (1) hæmatorrhachis or spinal meningeal hæmorrhage, and (2) hæmatomyelia or hæmorrhage into the cord substance.

(1) **Spinal meningeal hæmorrhage** is the most usual form, and may be *extradural* or *intradural*. Extradural hæmorrhage is the more usual disease.

Ætiology.—It is more common in men than in women. It also occurs in newly born children. Falls, injuries, and fractures of the spine are the most usual causes. Convulsive seizures due to epilepsy, tetanus, chorea, eclampsia, or strychnine may cause it, the same as in severe muscular strain. The various dyscrasiæ fol-

lowing infectious diseases, such as purpura, may cause it; or it may be due to the bursting of an adjacent aneurysm, such as of the aorta or vertebral artery, and in very rare instances cerebro-spinal meningitis.

Symptoms.—There may be no ascertainable symptoms in minute hæmorrhages. In large hæmorrhage there is sudden severe pain in the back, shooting down the limbs, associated with numbness, hyperæsthesia, muscular spasm, generally of the back muscles; later, paresis, paralysis, and anæsthesia may follow, with disorder of the visceral centres. The disease is fulminating, reaching its acme in the course of a few hours. Remission may then occur, accompanied by slow recovery or followed by chronic meningitis. Death may occur very early from exhaustion.

Diagnosis.—Injury, or childbirth followed by the sudden onset of attack, with irritative symptoms, as pain, which suddenly subside, point to extradural hæmorrhage. In hæmatomyelia there is less pain and irritation, but more marked paralysis and anæsthesia. This is also true of crush of the cord from fracture or dislocation. In tetanus the symptoms are more gradual in onset and trismus is present.

Prognosis.—If the patient survives beyond the fifth day there is a chance for partial or complete recovery. Usually the prognosis is most grave, the patient succumbing within a few days.

Treatment consists of absolute rest in bed, administration of sedatives and drugs to move the bowels and relieve pain. Local depletion may be tried, as by means of wet cups over the supposed site of lesion. The use of styptics, as suprarenal extract or mineral acids, may be of service in the purpuric cases. The sorbefacient effect of the iodides may be tried later; also the use of mercury. In chronic cases counter-irritation over the spine on alternate days for a fortnight may prove of value by its alterative action.

(2) **Hæmatomyelia.**—Hæmorrhage into the substance of the cord.

Ætiology.—This is not unusual. It may be due to vascular diseases or purpura hæmorrhagica, or may be secondary to tumours or to myelitis. Primary hæmorrhage occasionally occurs in infancy, but it is usually found in males in early adult life. According to Gowers, excessive coitus is a potent cause, as are injuries, exposure, overexertion, syphilitic endarteritis, or convulsive seizures,

which latter may also be the cause of hæmorrhage into the skin and intermuscular connective tissue. Hæmatomyelia may occur in old people with arteriosclerosis, spinal apoplexy being produced, instead of cerebral hæmorrhage.

Symptoms develop rapidly, with numbness or weakness for a few hours. Then sudden paraplegia, or ataxia, or both. Often the anæsthesia is dissociated—there being a loss of pain or thermic sense, with the retention of touch sense. The urine may have to be drawn on account of paralysis of the sphincters. The reflexes may at first be absent, but soon return and become exaggerated. There is much pain in the back, and if the lesion is high up the anus and thorax are involved. The acute symptoms will have subsided within a fortnight, when the case becomes practically one of chronic myelitis. If improvement has not occurred, the symptoms will instead take on the character of acute myelitis, and death will soon follow.

Pathology.—The blood-vessels diseased are those of the gray matter, which are in the ventral cord and under considerable tension. The rupture of these vessels is due to fatty degeneration or to specific endarteritis. Miliary aneurysms are rare in the cord, such as not infrequently develop in the brain. Hæmorrhage may be the cause or the result of a myelitis. In the former case the clot may be absorbed, leaving a cavity as in the cord ; or brokendown tissue may become the central foci of a myelitis. The hæmorrhage is usually single : it may be multiple. It is likely that cases of disseminated myelitis following infectious fevers are due to multiple ecchymoses. The bleeding at times is the result of new growth, as in syringomyelia.

Diagnosis.—Sudden onset without any long premonitory symptoms and the absence of fever followed by gradual improvement are characteristic of *spinal hæmorrhage.* There is much less pain than in *meningeal hæmorrhage,* while the dissociation of cutaneous sensation is very characteristic. In *acute softening* there is less of the dissociation of sensation, but a more extensive paralysis. The latter may be mistaken for acute primary myelitis, which does often follow. Meningeal hæmorrhage is usually more painful, and there is less paralysis, more spasm, and a more complete recovery afterward.

Prognosis.—This is often serious as regards life, and it is dubious as to recovery of power. Both much depend on the

extent and seat of the hæmorrhages. Dorsal hæmorrhages are more favourable, cervical the least.

Treatment.—Cold to the spine, such as through ice-bags, associated with absolute rest, together with small doses of cardiac depressants, as aconite, can be tried. Treatment must be prompt if any good is at all to be done. The after-treatment is entirely symptomatic, and later it is for the myelitis that may follow as a sequela (see Myelitis).

SYRINGOMYELIA

Is an affection of the spinal cord, which, while not more rare than disseminated sclerosis, has only been in recent years brought to full light. The disease was first accurately described by Schultze in 1886; since that time there have been some hundred cases reported in the literature, and in the light of the pathological findings undoubtedly many obscure cases of cord disease reported in the past have been of this nature.

There are two varieties of dilatation of the spinal cord: *First,* hydromyelia, which is a dilatation of the central canal, this being lined by the normal columnar epithelium. This condition may give rise to the symptoms of syringomyelia proper, similar to the *second* type, where the cavity occurs within the gray matter of the cord, but, as a rule, is separated from the central canal. A cavity or cavities in this instance are the result of softening areas in gliomatosis in the gray cord. The latter condition is probably hereditary, being an unnatural excess in development and continuance of the formation of this embryological tissue. The cavities may be *single, multiple,* or consist of numerous dilatations connected by irregular and deviating sinuses; so that one opening may be in this manner connected with another at a distance longitudinally from the first one. One-half the cord may be involved alone, or be at least more diseased than the other half. The cervico-dorsal cord is the most frequent site of the disease; although the medulla and pons, or lumbar and sacral cord, may be involved. That the disease has occurred following the infectious fevers does not particularly show them to be causative of the malady; so that the hereditary feature is the principal point in ætiology, as already indicated.

Symptomatology.—Four distinctive types of symptoms exist in certain groups of cases of syringomyelia, viz.:

1. The ordinary type described above.

2. Motor type, resembling amyotrophic lateral sclerosis.

3. Sensory type, in which disturbances of sensation, particularly loss of thermic sense due to disease in Gower's tract, are prominent features. It may be hemiplegic in distribution, or in irregular areas.

4. Those cases with pronounced trophic changes, painful whitlows, etc., as described by Schlesinger in 1895.

Fig. 59.—Atrophy of Intrinsic Muscles of the Hand and Thermo-Anæsthesia in a Case of Syringomyelia. (Philadelphia Hospital.)

The disease may resemble a number of chronic cord diseases in its clinical aspect, depending upon the impingement of the destructive lesion in various anatomico-physiological centres and tracts. Thus syringomyelia may resemble tabes of an irregular type when the posterior columns of the white matter are invaded, or it may present particularly the picture of progressive muscular atrophy of *spinal* origin if the dilatation occurs towards the anterior horns of the gray matter. In rare cases the disease may simulate spastic paralysis due to irritation of the lateral columns of the spinal cord; or in the rarest cases it may simulate insular sclerosis. So that given a case of chronic spinal cord disease of atypical or irregular manifestations, syringomyelia must be thought of. The particular symptom of diagnostic import is the dissociation of sensory phenomena—for example, there is usually loss of pain and temperature sense in irregular areas (analgesia and thermo-anæsthesia), with preservation of the sense of touch and muscle sense. If the cavity is on one side of the cord alone, and is of considerable size, we may have symptoms of Brown-Séquard paralysis, to be told from the usual type of " crossed

paralysis " of this sort by the absence of *pain* and *temperature sense* rather than the *touch* and *muscular sense*, as indicated; or as in spinal tumour or unilateral sclerosis, anæsthesia would be the sensory manifestation more particularly, or all forms of sensation would be absent. In cases where the cervical cord is affected high up the ascending branch of the fifth nerve may be affected, producing anæsthesia of the face. From involvement of the cervical sympathetic, the pupil on the affected side may be contracted, and there may be an absence of sweating on the same side of the face. Lordosis or scoliosis may occur in cases where the spinal centres governing the muscles of the spinal column are destroyed, this permitting muscular atrophies and consequent spinal curvature. In some cases motor cells of the spinal column are destroyed, permitting muscular atrophies and consequent spinal curvature. In many cases there is absence of the deep reflexes in the upper extremities; or if the disease *descends* low enough the knee-jerk may be absent— all depending upon destruction of the posterior roots of the spinal cord. The atrophy may extend to the lower extremities, and there may be reaction of degeneration found in the affected muscles. The course of the disease is slow, and may at any time come to a standstill. There may be remissions in the clinical course, no doubt due to extension of the process being abated. The patient may die from involvement of the cardio-respiratory centres in the medulla.

Treatment of this affection is of little value, although general hygienic measures to control the circulation by means of proper woollen clothing and the protection of the individual against inclement weather is to be instituted. Such patients should live an inactive life in the open air and sunshine. Continuous doses of iodide of potash may do good in the minority of cases through the alterative and sorbefacient effects of the drug. Re-education by exercises will prove of value where ataxia exists. Massage and static electricity may aid nutrition, better the circulation, and assist in staying the muscular wasting and sensory changes.

MUSCULAR DYSTROPHIES

These are classed under diseases of the nervous system, although no constant pathological finding has been found in the nervous system. Palsy is, however, a constant neurological symptom of all of them. There are three forms of muscular dystrophies, so called:

1. Pseudo-hypertrophic paralysis (Duchenne).
2. Idiopathic muscular atrophy.
3. Neuritic form of muscular atrophy (Charcot-Marie-Tooth).

(1) *Pseudo-hypertrophic Paralysis.*—The first, a disease beginning in early childhood, is a family type of disorder, although not known to be directly hereditary. The progenitors of the patient will be found to be alcoholics, subjects of syphilis, or some other form of degenerative disease. This has been satisfactorily proved to us in investigations made by Wharton Sinkler and the writer in a statistical study made in the Infirmary of Nervous Diseases of Philadelphia in 1899.[1]

Males are more frequently affected, and several in the family may be picked out, others escaping. The age limit of five to ten is the most usual time of onset. The patient first complains of feebleness in progression, and it will be noted that there is slight toe-drop, that the calves of the legs are enlarged, which greatly increase, causing a sense of firmness to palpation. Other points of pseudo-hypertrophy may exist in the pectoral muscles, the deltoids, and about the shoulder-girdle. With the enlargement comes on an increasing weakness of the muscles involved (Fig. 60). An early and characteristic feature is difficulty in arising from a supine position. The patient contorts the trunk in the endeavour to aid the specially weakened extremities, beginning by pushing the body up with the hands upon the thighs, then forcing the trunk to the vertical, thus extending the trunk upon the thighs, then drawing the lower extremities into the erect posture. The characteristic attitude is the hand-over-hand method in pushing up the trunk with the arms, in bringing the body to the erect position. This characteristic sign of the paresis may last for many months. There is no true ataxia, however. The patient is seen to have difficulty in mounting steps, and will frequently trip over slight

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[1] Journal of American Medical Association, November, 1899, Family Diseases.

elevations on the surface. Finally the patient cannot rise from
the sitting position, and it will then be found that the muscles,
which were apparently enlarged, have atrophied down to the nor-
mal size perhaps, or beyond this, and are board-like to the sense
of touch. Other muscles may pass directly into atrophy without
any pre-existent enlargement.

Reflexes.—The deep reflexes will be found lessened or absent
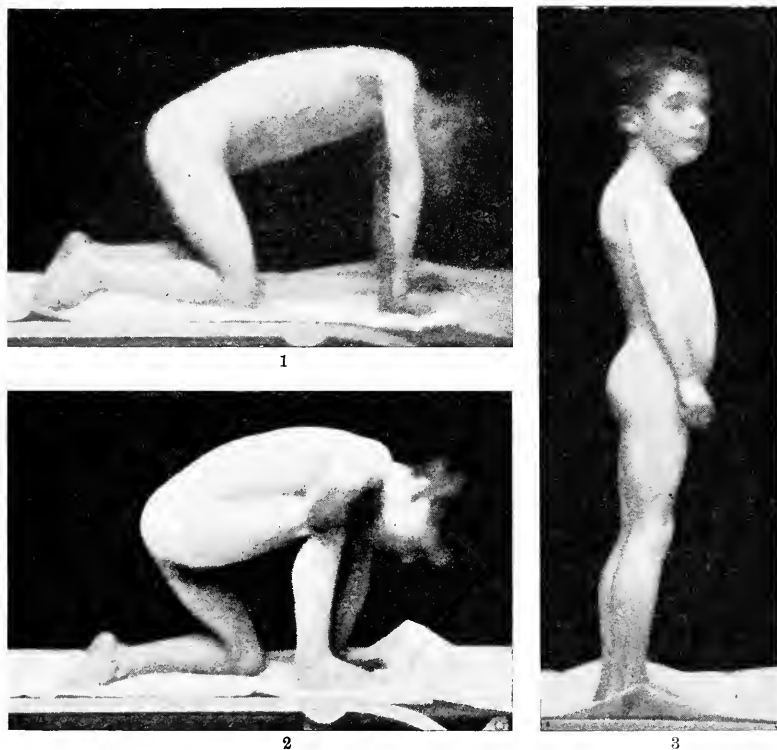
Fig. 60.—Pseudohypertrophic Paralysis showing Atrophy of Shoulder,
Girdle, and Thighs; also Manner of Rising in the Order 1, 2, 3.
(Medico-Chirurgical Hospital.)

and the superficial reflexes diminished. The end picture in this
malady is of extreme muscular atrophy and absolute helplessness.
Coldness of the extremities, cyanosis, enfeebled heart action, and
general lessening of the functionation of the special senses with
mental deterioration, occurs later in the affection, with dribbling
of saliva from mouth, difficulty in swallowing, etc. The average

duration of the disease is from one to twenty years, yet depending much upon the preserved nutrition and vegetative function of the patient; also upon his immunity or the absence of any of the complicating infectious diseases, which are particularly fatal in them. If not carried off by intercurrent disease, the patient may die of heart failure, from involvement of that organ.

Pathology.—This consists of excessive deposit of fat cells within the sarcolemma, and fatty change and swelling, loss of striation of the muscle cells. Overgrowth of connective tissue also takes place between the muscle cells. As the disease progresses the disappearance of the fat cells takes place, connective-tissue growth predominates, and the muscle substance is degenerated, leaving *débris* and connective tissue instead.

Diagnosis.—This should not be confounded with any other disease. In neuritis there is pain and tenderness of nerve trunks.

Prognosis.—An incurable malady. There may be prolonged remissions.

Treatment.—Much can be done to stay the progress of the dystrophy. This consists in persistent, scientific use of massage to favour better circulation, as well as of the elimination of the products of catabolism, and to prevent retrograde change in the muscle cells. Nutritious diet, consisting of eggs, red meats, or any other nutritious proteids, is indicated. Vegetables containing iron are valuable in this disease, in which metabolism is so perverted. In spinach iron can be introduced into the blood with least gastric disturbance. The overdistention of the stomach with carbohydrates is most certain to do harm. Predigested foods, as beef, is only called for when the stomach absolutely refuses to digest the more ordinary foodstuffs suggested. Electricity does not aid us much, although the static breeze may be of value in stimulating the circulation at the periphery, and to the carrying off of the waste products. The hypophosphites, phosphorus, and strychnine as a nerve tonic are all of value in this chronic disorder. Such patients should be carefully protected from cold and dampness, and given plenty of fresh air and sunlight, in a stimulating climate, at low altitudes. Sea air is particularly bracing.

(2) *Idiopathic muscular atrophy* is of two types: (*a*) the fascio-scapulo-humeral type (Landouzy-Déjérine), and (*b*) the juvenile form of Erb.

(*a*) In the former the atrophy begins in the orbicularis oris and
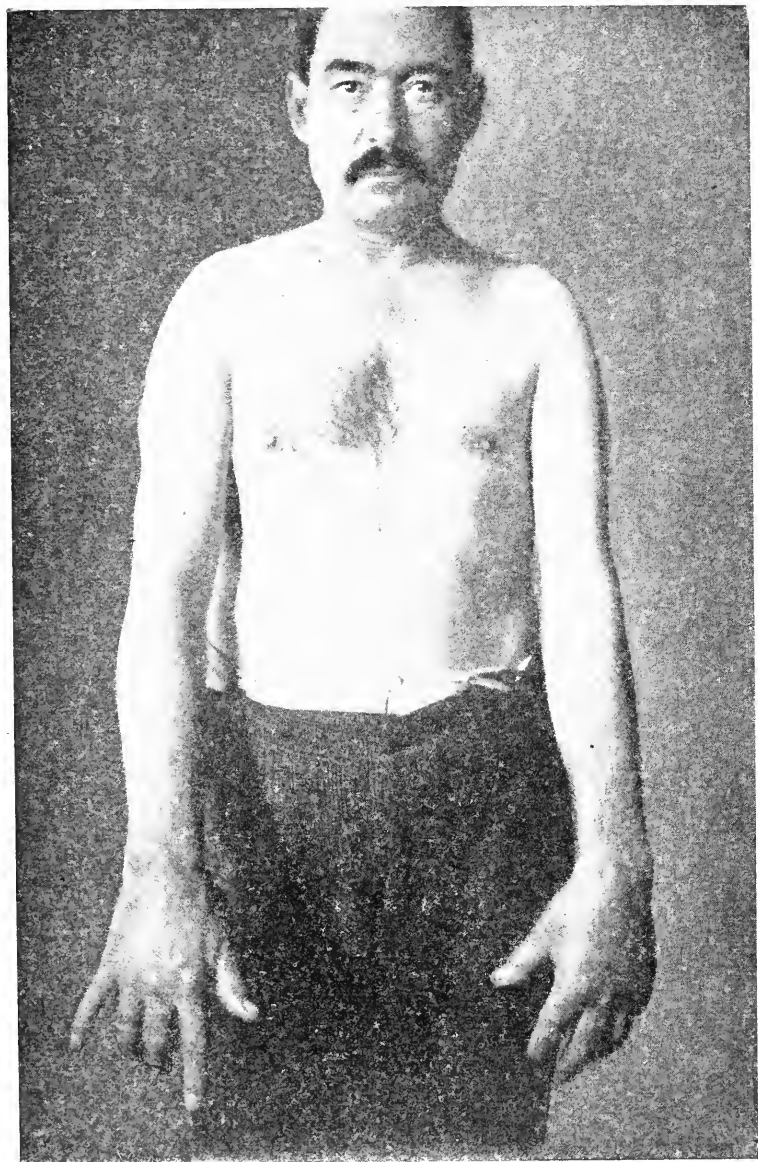
Fig. 61.—Progressive Muscular Atrophy of Spinal Origin (Anterior Poliomyelitis Chronica) showing Wrist-Drop, Atrophy of Hand Muscles, and of Shoulder Girdle, and the Pectoral Muscles. (Medico-Chirurgical Hospital.)

the face, producing the hollow-cheek appearance, the so-called "tapir mouth," and accompanied by fibrillary twitching of the muscles involved. This atrophy progresses in the trapezius and deltoid muscles, upper part of the arms, until within six months or a year there is a very marked physical change in the appearance of the upper portion of the body. The patient is unable to adduct or abduct the extremities, and particularly to raise the arms on a level with shoulder. Finally, the atrophy extends to the lower part of the arm and interosseous muscles, the lower extremities becoming involved last, if at all, in proportion with the duration of the case.

(b) The *juvenile* form begins in the interosseous muscles of the upper extremities, the thenar and hypothenar eminences, extending to the upper arm, finally to the lower extremities—thighs and legs—the patient's progression being with feebleness and evident toe-drop. This occurs most frequently between the ages of twelve and sixteen. It is very slow in progression, the patient living for many years. The *vaso-motor* weakness is shown by coldness and clamminess of the extremities, the indurated condition of the skin, brittle nails, etc. The deep reflexes are abolished early, but there is no reaction of degeneration, except in rare instances, very late in the course of the disease and where particularly large bundles of fibres are affected in the muscle atrophy.

The sub-type known as *progressive muscular atrophy of spinal origin* has a similar symptomatology, but begins in early adult life. This is the so-called *poliomyelitis chronica.* At times there is pain, the marked feature being fibrillary contractions, also that it occurs later in life than the muscular dystrophies proper, and that in the pathology of the disease, in addition to atrophy of the muscle cells and overgrowth of connective tissue, there is a degeneration of the multipolar cells in the anterior cornu of the gray matter of the cord.

(3) The so-called *neuritic type* is that which begins in the perineal muscles, producing peculiar guttering in the perineal region of the legs, with infrequently a toe-drop, and the process gradually extending up the thighs, and finally affecting the general musculature.

Pathology.—The pathology of this affection is not known further than the involvement of the muscles in atrophy and overgrowth of connective tissue.

Treatment.—In all these different forms of muscular atrophy it is largely symptomatic. Mechanical stimulation of the muscles directly by means of massage, Swedish movements, or galvanism are to be assiduously applied. Static electricity is a valuable agent to promote nutrition and bettered circulation in the tissues. The orthopædist should be consulted in many of these cases in order to prevent deformities as well as to strengthen joints enfeebled by the muscular disease, thus making life more useful in these patients, who might otherwise be prematurely crippled. The hypophosphites and cod-liver oil, plus a nutritious diet, consisting largely of proteids, with the use of eliminants, as copious draughts of water, the various mineral waters being particularly adaptable, are all agents of vital importance. Much can be done for these patients in the way of alleviation by persistent endeavour, even though cure is not established. Strychnine is of value in fairly large dosage—gr. $\frac{1}{20}$ to $\frac{1}{10}$ t. i. d.

ANTERIOR POLIOMYELITIS ACUTA OR INFANTILE PARALYSIS

This is a disease of sudden onset, more frequently seen in childhood, and is probably of microbic nature. The disease is fulminating, and is, as a rule, ushered in by a chill, followed by hyperpyrexia (104° F. or more), accompanied by nausea and vomiting and general restlessness. With these there may be hyperæsthesia and sensitiveness of one or more extremities. The fever lasts from several to forty-eight hours, as the case may be, when it will abate and the patient be left in a generally weakened condition, with palsy of one lower extremity, as a rule. Rapid wasting will supervene in the paralyzed part, and at the end of ten days or two weeks reaction of degeneration will be found in the paralyzed muscles. One lower extremity is generally involved, and the anterior tibial group more than other muscles, the next in frequency being the calf, the extensor femori, and the adductor muscles. Rarely an upper extremity is alone affected. There will be found an absence of the deep reflexes; as a rule the knee-jerk is wanting where the lower extremity is involved. The limb becomes cold, clammy, cyanosed, and wasted. If the member is not totally paralyzed, the patient will be able to limp about, and contractures, deformities of the limb, such as acquired clubfoot, occur later. The

disease very seldom extends to other parts of the cord after the
original attack; the patient generally recovers full and usual
health.   The paralyzed extremity does not grow in length as do
the others, and in consequence of the shortened limb there may be
a spinal curvature as a remote consequence.   The sphincters are
never involved in the acute attack of infantile paralysis.   Decubitus
seldom results.   The symptom of " palsy " is at first rather rapid
in its partial recovery, then promptly progressive.   In some cases
the deep reflexes are retained or increased.   It may occur in epi-
demic form in spring and summer.

Pathology.—This consists of acute inflammation of the multi-
polar cells in the anterior horns of the gray matter of the cord,
usually in the dorso-lumbar region.   Following the inflammation
there is found a degeneration of the multipolar cells, overgrowth
of connective tissue, and molecular *débris*; also degeneration of the
nerves coming from this particular segment of the cord, with
atrophy and fatty degeneration of the muscles supplied by them.

Diagnosis.—In neuritis there is tenderness of the nerve-trunks,
and as a rule it is less sudden in onset, with less severe palsy, though
a more general and constantly progressive or regressive paralysis.

Some cases of hip-joint disease may simulate infantile palsy
only in inability to move the extremity.   The hip-joint would be
partially ankylosed in coxalgia.   The pseudo-palsy of rickets is
always associated with that disease and there is little wasting and
no history of an " attack."   The knee-jerk is capricious.

Pseudo-muscular atrophy or other local forms of dystrophies
need but to be mentioned; as well as the cerebral palsies of child-
hood, which latter are associated with spasticity; while the former
are gradual in onset and widespread.

Prognosis.—This depends entirely upon the degree and extent
of involvement of the gray matter of the cord.   An attack very
seldom kills.   If a few muscle-bands are affected, it is possible that
almost complete recovery may occur with time and proper treat-
ment.   Where one member is markedly paralyzed usually consider-
able power will be restored within a few months' time, when fur-
ther recovery of the individual muscles will seldom take place.

Treatment.—This consists in the management of the attack,
which would be as of an ordinary acute infection, treating the
hyperpyrexia, giving a calomel purge, and protecting the affected
limb by carefully bandaging in cotton.   After the acute stage has

subsided, the general nutrition of the patient should be looked after. The use of the hypophosphites and a careful dietary are important, the remainder of the treatment being entirely local. This consists in massage and galvanism to the paralyzed muscles, continued for months or years, depending upon the severity of the case. If the child is able and there is no tendency to deformity, it can walk about; or if there be a tendency to deformity, the orthopædist should be consulted with the hope of " bracing " the weakened limb to prevent deformity that may occur in such cases. A high shoe may be necessary for the paralyzed limb where there is marked slowness of growth in it. The use of strychnine is of value, and should be given in doses according to the age, over prolonged periods of time. In inclement weather these patients should always be well clothed. They should be especially well protected against cold of winter and sequent frost-bite.

In cases where there is very little power remaining in an extremity, a method of assisting the exercise of the muscles is by daily immersing the child in a warm bath and permitting him to " float " the affected limb about in the water. The weight of the water displaced by the member will be just that amount of relief of dead weight in attempted exercising. I have seen great good from this method of treatment in one case.

GLOSSO-LABIO-PHARYNGEAL PALSY, OR BULBAR PARALYSIS

Bulbar paralysis applies to a number of systematized symptoms, due to diseases or lack of functionation in the centres in the bulb or oblongata (fifth, seventh, ninth, tenth, eleventh, and twelfth cranial nerves), and is divided into several varieties: (1) acute, (2) chronic, (3) asthenic, and (4) pseudo.

Symptoms.—These may accompany other diseases, such as amyotrophic lateral sclerosis, etc.

(1) The symptoms of *acute bulbar paralysis* are a sudden onset, dribbling of saliva, with the motor difficulty (dysarthria) of speech, and atrophy of the tongue. This acute form may be ushered in by an apoplectiform attack. It is also known as *polio-encephalitis inferior*. The symptoms are rapidly progressive, the patient losing the power to swallow. With the dysphagia there is loss of flesh. The patient is the subject of choking spells on attempting to swallow either solids or, in bad cases, liquids. In

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this affection the patient is perfectly conscious, but at times it is a difficult matter to determine whether the mind is affected or not, owing to the motor difficulty of speech. The latter is of a drawling nature, the words being prolonged, the consonants first being affected; finally the inability to pronounce words of more than one syllable with any degree of distinctness. The patient, however, is able to write perfectly well. With this added fact, bulbar paralysis is easily diagnosed from any form of aphasia.

The *course* of the disease is very rapid, and within three or four months' time the patient is in a very serious condition. Death is frequently caused by an inspiration pneumonia, or a sudden attack of choking; or from respiratory or heart failure, due to the involvement of the vagi centres.

(2) The *chronic* type of bulbar paralysis is much slower in its progress. The symptoms—stuttering, dysphagia, regurgitation of liquids from the nose on attempting to swallow—being *more gradual in progress.* The patient otherwise keeps in fairly good health, since the slowness of the process does not deprive him from getting ample nourishment until late in the course of the disease. The main difference between this and the *acute* form of the disease lies in the rapid progress of the latter, accompanied by general wasting, and the slower progress of the former.

The *pathology* of these forms is different in that in the acute types there is inflammation, a softening process, or minute hæmorrhages into the bulbo pons and cord, while the symptoms are apt to be consistently bilateral from the first; whereas in the chronic form, which is due to atrophy of the motor neurones, there is simply an aggression of the symptoms of one side out of proportion to the other, although both are involved. The *duration* of the chronic type is from eighteen months to three years. The patient may suffer from irregular cardiac action and attacks of tachycardia. It is somewhat singular that heart failure does not occur more frequently at the very onset of the disease, because of the involvement of the vagus centre.

The *prognosis* of both of the above forms is unpromising. Results of treatment may but ameliorate.

The *treatment* is more palliative than anything else, but considerable can be done in chronic cases to alleviate suffering by a careful attention to the manner of food administration, the use of the stomach-tube being a valuable method of feeding the

patient. This should be done at regular intervals during the course of the disease, even when the patient can swallow only a small amount, since in this there is liability to "inspiration" pneumonia. The use of strychnine in fairly large doses ($\frac{1}{30}$ to $\frac{1}{20}$ of a grain, t. i. d.) is of great value. The patient should live a very quiet life. Galvanism to the throat muscles is of value. Treatment of complications, such as pneumonia, is medical detail that need not be given here.

(3) The *asthenic* form of bulbar paralysis occurs later in life than the organic forms, and is more frequent in women than in men, occurring about the age of forty. It is designated asthenic because no definite lesion has as yet been determined, there being asthenia of the throat muscles.

The *pathology* is very like an auto-intoxication due to deficiency of metabolism, weakening the bulbar centres. The muscles supplied by the motor cranial nerves are involved; the centres are not able to generate or transmute nervous energy. Besides the symptoms already rehearsed other characteristic features of this form of disease are the remissions which recur in it regularly from time to time. Thus a patient may be unable to speak save in a whisper or in very indistinct monosyllables for a week or ten days, bodily weakness being present at the same time, and within a fortnight the voice may have returned, the patient having become much brighter mentally, and physically stronger.

Prognosis.—This is not good as to recovery, but the patient may live for many years in comparative comfort between the attacks, although harassed at frequent intervals by aggression of the symptoms. I have had the pleasure of observing a case of this sort with Dr. Wharton Sinkler at the Orthopædic Hospital for some three or four years.

(4) *Pseudo-bulbar palsy* is a type of progressive failure of the glosso-labio-pharyngeal muscles unassociated by atrophy of muscles or general weakness.

The lesion is very likely cerebral, affecting the upper motor neurones of the motor cranial nerves, particularly the ninth and tenth.

Treatment of pseudo and asthenic bulbar palsies does not differ from that of the chronic type. Good food, out-of-door life, arsenic, strychnine, the hypophosphites and glycerophosphates being of importance; also the use of gavage.

THE COMBINED SCLEROSES

By the combined scleroses is meant those forms of degeneration in which both the lateral and the posterior columns are affected. Westphal first described the condition in 1867. There are several types:

(1) Hereditary spinal ataxia (Friedreich's disease and hereditary ataxic paraplegia).

(2) Combined sclerosis in anæmic and toxic states (Putnam and Dana type).

(3) Combined sclerosis complicating general paresis.

(4) Accidental forms, as Gowers's ataxic paraplegia.

The clinical pictures of the many reported cases of combined scleroses vary greatly. Such cases must be of chronic ascending myelitis or meningomyelitis followed by degeneration. According to Marie and others, the vascular supply of the spinal cord is such as to favour sclerosis in the lateral and posterior columns, hence chronic leptomeningitis, many instances of which are due to syphilitic infection. The ataxic paraplegia described by Gowers, in which he believed the sclerosis lay in the lateral and posterior columns of the cord, is still observed as a clinical entity; but many similar cases can best be classed with tabes, with insular sclerosis, or with a form of limited chronic myelitis. We shall describe, therefore, only the hereditary form of ataxia with paraplegia, which is congenital and a family disease closely related to Friedreich's ataxia; also the type of combined sclerosis in which ataxia, some sensory and motor symptoms, are found associated with pernicious anæmia. There is no particular clinical picture attached to the cases of combined scleroses described by Strümpell and others with autopsies; hence the two above-indicated types at the present stage of our scientific knowledge, that are proper subjects for accurate description, clinically and pathologically, are (1) *hereditary ataxia* (see Friedreich's ataxia, p. 238) and (2) the *combined scleroses of anæmia*, etc.,as described by Lichthein, Putnam, and Dana.

(2) COMBINED SCLEROSIS OF THE TYPE OF PUTNAM-LICHTHEIN-DANA

This affection occurs past middle life, and is usually associated with cachexia, a pernicious anæmia, or some toxic states, as in influenza, lead poisoning, malaria, or chronic diarrhœa. Fre-

quently there is a history of heredity. This disease was described as developing in the course of anæmia by Lichthein in 1877; those produced by other causes by Putnam, and in 1891 again by Dana.

Symptoms. — The earliest symptoms are persistent paræsthesia, accompanied by slight weakness, most commonly in the feet, and somewhat later followed by ataxia. Pain in the limbs and back may exist. At first there is some spasticity of the muscles, increased knee-jerks, and ankle clonus. Later in the disease the rigidity may disappear and reflexes be lost in the lower extremities. After some months the arms become affected and symptoms develop similar to those in the legs. Occasionally the disease begins in the upper extremities.

Fig. 62.—COMBINED SCLEROSIS OF THE TYPE PUTNAM - LICHTHEIN - DANA FOLLOWED BY LEFT HEMIPARESIS. (Medico-Chirurgical Hospital.)

Lessening or absence of pain, tactile and thermic senses may occur, or there may be dissociated sensation, as in case of syringomyelia. In the course of the disease dementia may develop.

Pathology.—The posterior columns are first and mostly involved. In some cases of pernicious anæmia these were the only columns that were found to be sclerosed. In the majority of cases the lateral columns (crossed pyramidal tracts) are affected as well, and late in the course of the disease the anterior columns may be attacked. Softening of the cord with the production of cavities may appear. The condition is essentially a primary degeneration of the nerve-fibres.

Diagnosis.—This is to be distinguished from tabes. It is rapid in onset and there is some motor weakness, but absence of ocular symptoms is characteristic, while the early increase of knee-jerks with the anæmia should be taken into consideration. From ataxic paraplegia or posterior lateral sclerosis it is also told by its rapid development, the greater degree of weakness, and the constitutional symptoms. Those cases with dissociation of sensation are also distinguished from syringomyelia by the absence of severe pain and tenderness or of marked muscular wasting.

Prognosis.—This is unfavourable. In the majority of cases the patient will not survive longer than six months to several years, although cases of great improvement have been reported.

Treatment. — Hypernutrition, rest, massage, and Swedish movements. Measures tending strictly towards eradicating the diathetic or specific conditions underlying should be employed. The anæmia or any form of intoxication should be treated. The drugs of value in the disease are arsenic, quinine, iron, and other tonics, together with nutrients.

Re-education of the ataxic extremities can be had where improvement may happily occur if asthenia is not too great.

INFLAMMATIONS OF THE SPINAL CORD—MYELITIS

Myelitis, an inflammatory lesion of the spinal cord, is a disease rather insidious in onset, sometimes very abrupt, and running a course of several degrees; the *first*, or mildest, which frequently recovers, leaving little damage to the organic constituents of the cord; the *second*, running a serious course, and after subsiding the individual is a paraplegic; while the *third* type

is malignant, rapid destruction of the cord and death of the patient follows.

Causes.—These are exposure to cold and dampness; injury, such as a trauma to the back; or it may be of a septic nature, due to metastasis from other seats of infection, including tubercular.

Myelitis is much more frequently met with in the male, on account of the liability to exposure in men.

Pathology.—This condition consists of a low grade of inflammation with infiltration of round cells within the gray and white matter, more particularly of the gray matter. Granular *débris* is the result, plus fatty degeneration of the nerve-cells, and their replacement by connective-tissue elements. Where the process has been insidious and mild the neurones throughout the transverse section of the cord will be found but partially degenerated. In malignant cases there is a complete destruction of the parenchyma of the cord (see Infantile Palsy).

Depending upon the extent of lesion, we have *transverse, ascending,* and *descending* myelitis. The lesion of poliomyelitis need not be mentioned here.

Symptoms.—There is a rather sudden onset, a slight rise in temperature, paræsthesia and feebleness of the lower extremities. This continues more or less rapidly until the patient within a few days becomes bedridden, incontinence of urine follows, and perhaps of the rectal sphincter. The upper extremities may be involved, depending upon the site of inflammation, but usually not, since the process is generally confined to the lumbar cord. In the beginning the reflexes are heightened, ankle clonus even may develop, and the patient have a spastic gait and become more or less ataxic, depending upon the degree of involvement of the posterior white columns. Usually the gray matter is more involved, and if the multipolar cells are affected distinct atrophy and wasting of the muscles will follow. In a rapidly progressive myelitis, paræsthesia and hyperæsthesia (irritative symptoms) will within a few days to a week be replaced by a hypæsthesia or anæsthesia, involving both lower extremities very likely in their extent, or the trunk may be thus affected as high up as the umbilicus. At this stage bladder distress from incontinence and paresis of the detrusor muscle will permit of retention of urine as well, and resulting cystitis. At this time, too, bedsores may occur, particularly liable over the buttocks and sacral region, due to the low-

ered trophic condition and pressure upon these dependent parts. At this last stage the reflexes will be abolished. The patient is restless, loses flesh, and becomes hectic, and may go into a septicæmia, which may cause death; or after a number of weeks the inflammation may subside and the paresis partly recover, permitting the patient to get about on crutches within a few months to a year. In other cases the destruction of the cord may be so absolute that even partial recovery of function seldom occurs, although held in abeyance, and the patient may go on living a vegetative life for some months or years. This is especially apt to result in those cases of traumatic origin, such as in " broken back " of the coal miner.

In some few cases I have seen almost entire recovery after a most severe attack, and in these cases it is very likely that the meninges have been particularly involved, and while the function of the cord for the time is greatly interfered with, the absolute destruction of the neurones has not occurred. The extremities involved in myelitis become cold, clammy, cyanosed, etc. On beginning recovery of a case the patient moves about with a spastic gait, which he will retain in some measure, although he may pretty completely recover from the palsy.

Diagnosis.—This is to be made from neuritis, where pain and tenderness along nerve-trunks would differentiate; from subacute rheumatism of the limbs, which would be distinguished by the nature and distribution (to the joints) of the pain with other symptoms of the rheumatic disorder; and finally from syphilis of the spinal cord, where the course of the disease is much slower, and spasticity is out of proportion to palsy. In syphilis too seldom are the signs equally bilateral; paresis is less marked and muscle tone not increasing in proportion to the heightened reflexes existing, as indicated.

Prognosis.—This is a very difficult matter to determine. It should be held in abeyance until the first few weeks of the disease have passed, and will depend in some measure upon the physical stamina of the patient, although distinctly plethoric persons are apt to suffer the greatest. Infections cases particularly will often recover more completely than the so-called idiopathic class. The tubercular cases are the most promising for restoration of the function of the cord. Here, again, the inflammation is liable to be localized in the membranes.

Treatment.—This consists in absolute rest in bed, preferably a water-bed; the use of mild antipyretics, if the case is fulminating in type, and there is much constitutional involvement. Protection should be had to the extremities by means of " hoops " to sustain the bed-clothing, and the dependent part should be amply guarded against prolonged pressure by frequent changes of the position of the patient. The use of analgesics may be necessary (phenacetine, 5 grains) where there is considerable distress. The bladder should be guarded, especially for retention of urine, by a careful use of the catheter, both to prevent injury of the mucous membrane and from the aseptic point of view. If marked cystitis develops it may be necessary to flush the bladder with some antiseptic solution, as boracic acid or a minimum solution of potassium permanganate (1 grain to a quart of water). The decubitus developing should be antiseptically cleansed, and a soap plaster placed around the adjacent tissues to prevent pressure on the sore, the latter being antiseptically treated. The so-called " ring," when used, I have not seen of practical value, since it disturbs the patient, and will slip and only do more damage in the already inflamed area. Among drugs, the solution of potassium iodide, directed to be taken in 5- to 10-grain doses, t. i. d., is of great value from its sorbefacient effect upon the exudate about and in the cord substance. This should not be pushed to a high dosage, as in cases of specific disease, which shall be mentioned in the next chapter. Prolonged rest is essential for the hastening of good results. The necessity for care of the skin in any case is to be insisted upon by the proper bathing and stimulation with alcohol, etc. Thorough massage is a valuable aid in restoration of tone of the muscles in convalescing cases, and should be continued for months. When the patient gets about he should be cautioned to use crutches or a cane and to avoid activity, lest he increase the inflammatory process.

The treatment for myelitis following Pott's disease differs in one particular—i. e., in the use of *extension and counter-extension* to the patient by means of the jury-mast, which is weighted from the head of the bed, the counter-extension being made by elevation of the head of the bed. This extension may be increased to from 10 to 30 pounds, and should be practically continuously applied to the patient until the inflammation of the vertebræ subsides and the palsy has partially recovered. This class of cases comes under the neurologist's care when the paraplegia is marked, and may

reach the orthopædist when the vertebral symptoms predominate. Another method of extension is by *suspension* of the patient while sitting in a chair by means of an inverted " L "-shaped rod attached to the back of the chair, to the short end of which a pulley, scales, and the head-extension apparatus is attached. This can be nicely adjusted to the weight desired for the extension. I have seen patients with complete motor and sensory palsy of three months' duration recover entirely from palsy, by combination of the bed-and-chair extension described, within seven months of persistent treatment. The tendency in these cases to relapse is to be noted, and a brace should always be worn afterward to support the spinal column. Cases of Pott's disease with suppuration are least apt to develop paralysis, very likely because extension is outward rather than inward in pus cases; also due to the apparent immunity of nervous tissue to sepsis.

CHAPTER XV

DIFFUSE DISEASES OF BRAIN AND CORD

Multiple sclerosis, or **insular sclerosis,** or disseminated sclerosis, or *sclérose en plaques,* is a chronic affection characterized by focal areas of sclerosis scattered throughout the brain and cord; occasionally the sclerosis is limited to the brain or to the cord.

Ætiology.—Multiple sclerosis may be the result of trauma, as of the spine, to the influence of exposure to cold and wet when various metabolites and toxins are developed in the blood as sources of irritation. Overwork may in the same way act as an exciting cause, as will emotional states, which may also much pervert all bodily secretions and excretions. Infectious diseases or the metallic poisons, such as of lead or mercury, may also act as causes.

Symptoms.—The symptoms will vary in accordance with the distribution of the sclerosis as well as its extent. Still there are certain signs and symptoms of sufficient importance to be characteristic. These consist of weakness of one or more extremities, such as of the legs. The limbs become spastic, the deep reflexes being increased. A coarse tremor develops in the affected parts, and this is of the intention type—i. e., it is awakened or exaggerated by volition or a voluntary act. These irregular tremors may involve the arms, legs, and finally the head. The tremor usually subsides when the parts are at rest, and absolutely so when the patient sleeps. Occasionally the tremor is so coarse that it is difficult to differentiate it from ataxic movements. Nystagmus is a common symptom. This may be persistent, and is usually *lateral nystagmus.* It may, however, only be brought out by having the patient strain the eye muscles, as in looking far to one side in attempting to produce conjugate ·deviation. Ophthalmoplegia may finally occur. Optic atrophy is frequently found, but the Argyll-Robertson pupil seldom occurs. The lower cranial nerves, such as the fifth, seventh, and twelfth, may be also involved. **Speech** is apt to be affected; when it is slow and sing-song with

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elision of letters and words. The patient articulates as in scanning poetry, the so-called *staccato* utterance or *scanning* speech. Sometimes atypical forms of dysarthria, resembling the speech in bulbar palsy, or Friedreich's ataxia, or even paretic dementia, may be seen. At times patients affected with insular sclerosis may be subject to attacks of vertigo, apoplectiform or epileptiform seizures. In unusual cases anæsthesia may be existent, when it occurs in the form of small irregular patches, although if a sensory tract is involved and destroyed high up hemianæsthesia may result. Of course various forms of paræsthesia may be found, since irritation of sensory tracts without destruction of them is the more frequent condition present in the brain or cord in this disease.

Diagnosis.—Most of the cardinal symptoms just enumerated will at once serve to differentiate this malady from any form of *system disease*, which latter would only be simulated where there is a large plaque involving a special tract, as the sensory, for example, as mentioned above. The symptoms may remain entirely cerebral or spinal. Remissions are frequent.

Intention tremor, scanning speech, and nystagmus are rare in *cerebro-spinal syphilis*; while in syphilis, too, there may be iridoplegia in one or both eyes, or the pupils may be irregular in contour from previous synechiæ, which latter would never occur in insular sclerosis (Sachs). The tremor of *paralysis agitans* is more marked when the part is at rest, and it is inhibited by volition or voluntary movements—a dynamic or continuous tremor. Parkinson's disease is also one of old age, and true scanning speech or nystagmus are neither present in it. Paraplegic forms of *infantile cerebral palsies* are told by their appearance in very early life, occurrence of convulsions, associated with a more pronounced failure of mental development. The diplegic type is more difficult to distinguish, since *scanning speech, nystagmus,* and *ataxic movements* may be present. The syndrome of all three of these last-mentioned symptoms is, however, seldom found in cerebral palsy; also paralysis and contractures are apt to be marked in cerebral palsy. In the *hereditary* ataxia of Friedreich, optic atrophy, lessened or absent reflexes and ataxia of lower limbs are points in diagnosis. From the *cerebellar* type of hereditary ataxia it is more difficult to distinguish. Romberg's sign, which is present in cerebellar ataxia, is, however, very rare in multiple sclerosis.

Prognosis.—Insular sclerosis is slow in progress, but is incur-

able. Remissions may occur spontaneously or may be due to treatment in those patients with good physiques.

Treatment.—Alterative remedies, such as arsenic or Donovan's solution, or even the iodides, may be of much service. Nitrate of silver has been commended, but I have never seen benefit from its use. Re-education by teaching the patient to co-ordinate is of value in the symptomatic management of the case. Massage is important. General nutrition of the patient (such as through the administration of syr. ferri iodidi, gtt. xv, t. i. d., or cod-liver oil ℥ss. after meals) is the most important part of treatment.

PARALYSIS AGITANS (SHAKING PALSY)

Paralysis agitans is a disease occurring more frequently about the fifth decade of life, and in which tremor and palsy are features. The name *Parkinson's disease* is also applied.

Causes. — Heredity, overwork, or trauma to spine.

Symptoms.—The cardinal features are paresis, accompanied by fine constant tremor, beginning as a rule in the hands, face, and

Fig. 63. — Expression, "Pill-rolling" Hands, and Attitude in Paralysis Agitans (Adult Male; but little Tremor). (Philadelphia Hospital.)

lips, gradually spreading until the entire musculature is involved. The tremor is a *continuous* one in the majority of cases when the disease has become well established, although there is the unusually rare case that does not manifest tremor except periodically. The

tremor is never what we designate an intention tremor—i. e., one brought out by voluntary movement of the parts; in this we have a diagnostic symptom, as between it and insular sclerosis, in which there is generally marked *intention* tremor. Other signs of paralysis agitans are a masked expression, the so-called " wooden face," due to rigidity and immobility of the muscles of expression; and increased reflexes. With these there is slowness of cerebration, and still more of articulation due to mechanical interference with speech from lingual and pharyngeal involvement in the muscular rigidity and paresis. Later, as the disease slowly advances, dribbling of saliva, very marked stooping of the body with rigidity of the neck, an assumption of the pill-rolling position of the hands and fingers are characteristic, and finally the *festinating gait*, or running gait as it is sometimes called. This gait is really an exaggeration of that of old age, and in which the patient is said to be running after his centre of gravity (not an inapt term). If the subject feels himself falling forward, the term *propulsion* is implied; if he has the sense of falling backward, we designate it *retropulsion*. The circulation is generally feeble in these patients, cardiac palpitation is not uncommon upon slight exertion, there is usually marked arteriosclerosis, and frequently iridoplegia in myosis. The tremor in its incipiency is also more marked when the patient is relaxed, though it is exaggerated with the relaxation following extraordinary exertion for him; and finally tremor may become so large and persistent that sleep is prevented from the mere mechanical effects upon the body, but when the patient does sleep the tremor disappears for the time being. Save for paræsthesia no *sensory symptoms* are found, excepting in rare cases, where there are rheumatic or articular changes of a trophic nature in the joints, when fleeting arthritic pains may occur. The patient is eventually compelled to give up the use of the hands, as in writing first, then of larger movements; although it is singular, the ability to write is preserved to extreme advance of the disease in some cases. Local flushing of the face and neck and body with evanescent sweating of the parts is always a notable symptom in this disease, due to vaso-motor irritability. The sphincter muscles are almost never incontinent.

The *duration* of paralysis agitans is indefinite. As the disease is incurable in itself and occurs at the degenerative period of life, the demise of the patient will depend upon the complication of

other maladies, or upon the degree of degenerative tendency in the individual. The latter should be sought for in some measure by noting the heredity of the family.

Pathology.—While no lesion has been found in the majority of cases coming to necropsy, in a fair proportion of them changes in the cord have been noted, largely consisting of congestion and dilatation of blood-vessels of the gray matter, atrophy and pigmentation of nerve cells, and increase of the interstitial tissue. Dana considers, however, that the central motor neurone is most at fault.

Prognosis.—As inferred from the above, recovery never occurs; but taken early, the malady can be controlled very materially in 25 per cent of the cases. Remissions and exacerbations are notable. I have in mind a patient, who will be referred to under treatment, in whom there has been very marked amelioration of the symptoms, especially in the restoration of general tone to the system, although the tremor has never been greatly improved. Another advanced case at the Philadelphia Hospital under my care has been greatly improved as to the tremor itself by therapeutic measures. Where it is clearly shown that there is no family tendency to the disease we can hope for better results than in cases with neurotic taint in a preceding generation. Just when the stage of paresis will develop cannot be accurately foretold, although generally speaking, the greater extent and range of tremor the earlier will palsy result.

Treatment.—The *essentials* in treating paralysis agitans are rest and hypernutrition. In an incipient case, therefore, ideal treatment lies in change of scene, the leading of a quiet life, with abundance of fresh air and sunlight. Light gymnastics and passive movements are of value in preventing rigidity of the limbs.

Tonics, as quinine, arsenic, and mineral acids, are helpful. Warm baths daily and the constant galvanic current are of value. Hyoscine hydrobromate, gr. $\frac{1}{100}$ t. i. d., will frequently relieve the symptom tremor, or a combination of tincture of conium and tincture of hyoscyamus, equal parts, given in 5- to 10-drop doses, is of service. Gowers recommends cannabis indica and arsenic. I have seen the Brown-Séquard [1] injections do good. Elimination through skin, kidneys, and intestinal tract must be regulated.

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[1] Testicles of healthy bulls are ligated and excised immediately after killing the animal. Under antiseptic precautions the tunics are completely

SYPHILIS OF THE NERVOUS SYSTEM

General Considerations and Distinctions

Scientifically speaking, "syphilis of the nervous system" applies to the *tertiary* lesion located in the membranes of the brain or cord, or both; although *parasyphilitic* disease or the toxin stage (or quarternary stage of some authors) is also a sequela of syphilis. In the latter, however (as in tabes and paresis), there are no characteristic lesions of syphilis. In it, too, the specific poison may expend itself upon the progeny, affect the general health, lessening resistance, and may create morbid tendencies or even sclerosis, as inferred above, in descendants of parasyphilitics.

The acquired syphilitic diseases of this parasyphilitic stage may therefore be paresis, ataxia, neurasthenia, hysteria, epilepsy, and mental diseases. The congenital syphilitic diseases of this same stage may be arrest of development (as in Little's disease), hydrocephalus, paresis, juvenile tabes, the hereditary form of cerebral palsies in children, etc. We have described all these various parasyphilitic diseases in other chapters.

NERVOUS SYPHILIS

General Considerations.—In the so-styled *specific* form of syphilitic disease of the central nervous system there may be the *hereditary* or *acquired* forms. In the former, symptoms are present at birth or may appear early in childhood. Very rarely this form may even appear late in life, affecting all parts of the nervous system singly or collectively. The *acquired* and usual form may appear at various times after the primary lesion. Usually symptoms present between eighteen months and six years. Some acute fulminating cases may even occur during the secondary period. Others may, exceptionally, occur even after thirty years of the luetic infection.

Pathology.—There are various forms of tertiary lesion found.

(1) *Disease of Blood-Vessels.*—*Endarteritis obliterans* is frequently found in which there is proliferation of endothelial and

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removed down to and including the albuginea. The gland is then macerated in pure glycerin and allowed to dissolve for forty-eight hours. The whole is then filtered and a small extra amount of the liquid part of the residue is added to make a supersaturated solution. The mixture is then sealed in antiseptic phials. By this method one testicle is equal to one ounce of solution.

subendothelial cells. This much thickens the walls of the blood-vessels, and narrows and sometimes obliterates them completely. The other coats may also be destroyed. Periarteritis in which nodular gummata may develop in the adventitia of the vessels may produce globular or ovoid swellings. The interna may or may not become diseased. Secondary thrombosis, softening, or rupture or the development of aneurysms may occur from any of these blood-vessel lesions. Cases where the secondary symptoms have been mild are particularly apt to develop syphilis of the nervous system. This is probably due to insufficiently treated specific disease or to a vulnerable nervous system, or both.

(2) *Gummata.*—These may be single or multiple, and usually involve the pia mater, affecting the brain or cord secondarily, frequently only by compression. They spring from the walls of the blood-vessels, and are composed of round cells derived from them and from proliferated connective-tissue cells. Hence the walls of the blood-vessels become thickened. A gummatous meningitis also is usually found. The gummata may be attached to the dura, and rarely to a vessel of the brain or cord substance proper. Gummata are not as commonly *found* in the cord as in the brain. In the brain they are usually seen upon the cortex or in the pons (hence the symptoms of palsy will depend on location of growths).

(3) *Gummatous Meningitis.*—This is a condition in which the membranes are infiltrated with cells, and are much thickened in consequence. This is found always about gummatous masses, but may exist separately. It is more common at the base of the brain, and generally involves the dura. Gummatous growth themselves are much less frequent at the base than is specific meningitis.

Symptoms of Cerebral Syphilis.—The onset of symptoms is generally from three to six years after the initial specific lesion or chancre. The patient will complain of drowsiness in the daytime and wakefulness at night, with persistent headache, due to meningeal irritation and inflammation. According to H. C. Wood, he becomes a restless nocturnal automaton. The mental conditions of obtundity, with failure of memory, but without delusions, are other distinctly psychic phenomena. Ocular palsies are very likely to develop early, perhaps the first symptom being that the patient will complain of seeing double (diplopia) ; or it may be noted that he has a strabismus, or on testing the ocular balance that the movements are restricted, due to neuritis of the ocular motor nerves.

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because basilar exudate is common. The patient is physically weak, the reflexes are heightened, due to the chronic irritation of the motor tracts and centres; and localized palsies may supervene in other places, such as in an arm or a lower extremity, due to destruction or pressure upon the motor centres. Acute optic neuritis and optic atrophy may develop, but is uncommon, while " choked disk " is but seldom seen, thus differing from eye symptoms of brain tumour. The patient may develop convulsions from time to time in proportion as the process is chronic. Various paræsthesiæ of the extremities may occur in cerebral syphilis. A palsy is almost never complete in this disease, and the vacillation from month to month is a point in favour of this diagnosis. One of the principal points in the symptomatology of cerebral syphilis, we wish to insist, is early palsy of the ocular or other cranial nerves. Such palsy also varies much in extent from week to week, according as active specific treatment is pursued or not.

Symptoms of Spinal Syphilis.—While the symptoms are usually conjoined with those of cerebral syphilis, still the predominance of the spinal disease occurs in a sufficient number of cases to warrant the title given, and which has frequently been called, since Erb first well described it, *Erb's paralysis.* The time of onset of spinal syphilis is the same as that of cerebral syphilis (usually two to four years after infection). An early complaint is of paræsthesia of the lower extremities, accompanied by feebleness and a sense of rigidity of the muscles, with at the same time incontinence of urine or difficulty of urination, due to weakness of the vesical sphincter. Indeed bladder symptoms are the earliest symptoms of the disease. The reflexes are heightened, the knee-jerks being greatly increased, and marked sustained ankle clonus occurs. Seldom is there anæsthesia, but hyperæsthesia or hypæsthesia frequently exists of irregular distribution, depending upon the segment of the cord and roots involved. Some disturbance of co-ordination and Romberg's symptom may be present. A clinical point of particular note is the *heightened reflexes out of proportion to palsy and the associated proportionate diminution of muscular tone;* so that seldom do contractures occur, even though the knee-jerk is wildly spastic. The muscle development remains good; there is seldom wasting or fibrillary contractions, but cyanosis and coldness of extremities among vaso-motor phenomena are frequently seen. Bedsores or other severe trophic changes are rare,

because all nerve-paths are seldom entirely destroyed. The non-symmetrical distribution of symptoms is also a special clinical feature in syphilis of the spinal cord, as is also the *remission* of the symptoms.

The *duration* is indefinite, and tends towards degeneration or sclerosis if not properly treated. The patient is liable to sudden death, though usually the course is slow.

Diagnosis.—Careful observation should not confound this disease with any other malady. Confusion may be had with myelitis, where the symptoms are symmetrical in distribution, seldom with remission, while the onset is acute in character and accompanied by bed-sores. There are also distinct sensory symptoms, such as girdle sense and anæsthesiæ, throughout the course of myelitis.

Prognosis of Cerebro-Spinal Syphilis.—It is most hopeful of all organic affections of the central nervous system, excepting in the fulminating cases, where the profound intoxication destroys the nerve-cells before relief can be had through timely treatment. These latter cases are the rarest and do not present the typical features described above.

Treatment.—This consists in vigorous and thorough application of antisyphilitic remedies. The use of potassium iodide, increased to 100 or 200 grains a day (taken in solution), will prove of greatest value. It is astonishing to see the good results obtained in the cases that have received prompt and early treatment. Where the iodide cannot be borne, or in case there be no response to the drug, the use of bichloride of mercury in $\frac{1}{24}$-grain doses, rapidly increased, is a therapeutic procedure of great value. Inunctions of mercurial ointment into the axilla and groins, on alternate days, will also be efficacious. During the administration of antisyphilitic measures the efficacy of the drug action may be bettered by the use of hot steam (Russian) baths given once or twice weekly. The stomach must be conserved in all cases, and if gastric symptoms occur the syphilitic treatment should be held in abeyance for a time and gastric sedatives and tonics administered, after which the specific treatment may again be resorted to. Reconstructives are essential, such as cod-liver oil, phosphorus, and hypophosphites. Copious draughts of water should be given, and the bowels should be kept freely open and the contents soluble by means of salines. The diet should be of a simple, wholesome nature, such as eggs, milk, and other proteid food

in limited measure. Careful treatment of syphilis of the nervous system, as outlined above, persisted in for six months' time at least, will often produce excellent results; and some cases seem to be restored to normal health. If improvement does not occur within six months degeneration of the nerve-cells has most likely set in, and a favourable issue cannot then be expected, although treatment should be continued with the idea of ameliorating the dire effects of the syphilitic poison or toxin itself.

CHAPTER XVI

GENERAL AND FUNCTIONAL NERVOUS DISEASES

IN functional nervous diseases no definite known anatomical change underlies the morbid alteration of function. We therefore classify such affections *clinically*. There may be made, however, *pathogenic* and *etiological* classifications. Thus we may have primary or degenerative and secondary or acquired neuroses. This classification is helpful, though not perfectly correct, because several factors often enter into the cause of the same disease. Classification should not be made a fetich.

Dana gives the following classification, which serves well in the majority of diseases of this nature:

Primary degenerative neuroses...
- Primary neurasthenia, hypochondriasis.
- Epilepsy.
- Hysteria major.
- Hereditary chorea.
- General spasmodic tics.
- Myotonia.

Exhaustion and shock neuroses.
- Neurasthenia.
- Hysteria.
- Exophthalmic goître.
- Occupation neuroses.

Acquired neuroses.
- Acquired degenerative neuroses.
  - Tic douloureux.
  - Local spasmodic tics.
  - Paralysis agitans.
- From infectious, autochthonous, and mineral poisons.
  - Chorea.
  - Tetanus.
  - Tetany.
  - Rabies.
  - Tremor and neuralgia.
- Miscellaneous.
  - Vaso-motor, trophic, and sleep disorders. (See appendix.)

We shall give the most important of the general and functional diseases of the nervous system, classifying them clinically, but

with the understanding that the cause of the disease is always borne in mind to be of first importance in considering its treatment.

CHOREA MINOR, SYDENHAM'S CHOREA OR ST. VITUS'S DANCE

Chorea is a general disease, at present designated functional, since its ætiology is not definitely decided. An autochthonous poison, as in rheumatism or other diathetic disease, may be the exciting cause; or it may possibly be infectious in origin. Usual exciting causes are mental, moral, or physical trauma (fright, blow upon the head, etc.). Anæmia and general ill health and heredity are predisposing factors.

Definition.—Chorea is a functional nervous disease associated with irregular inco-ordinate movements of the voluntary muscles, and accompanied by mental obtundity, anæmia, and at times cardiac and rheumatic symptoms.

Paralytic chorea is the name applied to that form of St. Vitus's dance where muscle palsy from exhaustion is markedly present. There is more or less paresis in all cases.

Symptoms.—Sex bears important relation to the disease, it being found much more frequently in the female; perhaps the proportion is about two to one. Onset occurs most frequently between the seventh and fourteenth years. The adolescent period has something to do with the development of the cases occurring latest in this susceptible epoch.

The first symptom is generally peevishness of the child, accompanied by general restlessness, which the mother will describe as "fidgets." The child will drop an object without any apparent cause—e. g., a fork at the table, or the pencil if working at school. Within a week the movements will have begun in the arms, face, tongue, and lower extremities in the typical case. The movements are very irregular, in some cases tending to vermicular motion, but never in any degree consistently of one type. The peculiar contortions of the face give the child a grotesque appearance, which with the difficulty of speech and dribbling of the saliva present the aspect of a case of dementia (facies choreica). This is at once unwarranted by the mental condition of the case to be described later.

The gyrations become larger and increased until the range of

movement may extend to extreme abduction of the extremities, the throwing about of which will prevent the child from walking or even sitting erect from mere mechanical disturbance. In other cases the palsy is so much greater than the movements that they are designated the " paretic type." In these the patient is at times absolutely disabled, with a few movements remaining. In other cases the irregular movements appear well marked and yet are associated with palsy out of proportion to the motor weakness.

The movements of chorea cease during sleep. They are much bettered when the child is quiet, and are exaggerated on physical or mental endeavour. At times chorea is confined to one side almost exclusively, which type is termed *hemichorea*, or one side of the body may be affected more than the other. Occasionally one upper extremity and the opposite lower will be most involved.

The child's mental condition is below par, and while sensitive, the function of the mind is singularly obtunded, memory being poor, with inability to concentrate. In some cases there is mild ephemeral dementia ; in other cases the dementia may go on to permanent mental impairment or death. In rare instances an excitable condition of insanity without high temperature will occur.

S. Weir Mitchell divides chorea into five types, as follows: (1) Those with absence of movements during rest, during some stage of the disease. (2) Cases in which continued movements are increased by effort. (3) Cases in which movements disappear during intentional muscular acts. (4) Cases unaltered during muscular acts. (5) Those in which the various types alternate.

In violent cases the child may injure himself by tossing about, so that restraint is necessary. The only somatic symptom of importance is feeble and irregular cardiac action. In 75 per cent of cases a systolic mitral murmur occurs, either hæmic or due to a mild grade of endocarditis.

The *duration* of a case of chorea is from four to six weeks. Severer cases may last many weeks. Recurrences are quite frequent, many cases having relapse in the spring months (March and April), or in the early fall (September). This is probably due to the effect of the changing weather as well as to overwork at those periods of the year. The subsequent attacks are generally not as long as the primary ones, and may recur from two to fourteen times in as many years.

As the patient convalesces from the attack the movements grad-
ually cease. Frequently when the paresis is very prominent the
child complains of greater weakness and apathy than when in the
attack. The movements cease last in the face and tongue, physio-
logically the most sensitive parts of the body. Extreme anæmia
will at times develop, a cardiac murmur at the base be very pro-
nounced, and even fatty degeneration of the heart muscle be shown
by the feeble contraction of the myocardia. In some cases œdema
of the lower extremities will be noted. The urine is generally de-
ficient in chlorides, with excess of sulphates and phosphates, show-
ing the waste in the nervous system; while indicanuria is a fre-
quent finding in those cases where gastro-intestinal fermentation
exists, as a complicating factor in symptomatology through auto-
intoxication. In rheumatic cases the mitral cardiac murmur may
be very persistent, due to organic disease of the heart, which symp-
tom may dominate in the case, and be the sequel of an apparently
mild attack of chorea. The co-relation of rheumatism, chorea, and
heart disease is therefore to be borne in mind; while a patient suf-
fering from rheumatic pains with cardiac disease before or in the
attack of chorea is a more gravely ill person than one not having
this trio of signs. The knee-jerk is capricious in chorea, fre-
quently lessened, and always easily exhausted.

Diagnosis.—Chorea minor is not likely to be mistaken for
other diseases. *Hysteria* may simulate this disorder, but the ex-
plosive nature of hysterical chorea, the larger movements, and
less palsy will distinguish it. *Habit chorea* is told by the spas-
modic and localized character of the movements. The choreiform
movements of the *cerebral palsies* are to be differentiated by noting
the other signs in the disease in question, such as palsies, increased
reflexes, etc. *Huntingdon's* chorea is differentiated by its occur-
rence in adult life and its association with mental diseases; also
the hereditary nature of Huntingdon's disease.

Prognosis.—The prognosis of chorea is good in the vast ma-
jority of cases. A usual duration is six or eight weeks. The
cases with cardiac affection are more apt to be prolonged, as shown
by murmur, even if the heart disease is functional; also the anæmic
cases are graver, due to the condition of malnutrition of the cen-
tral nervous system in these patients. The prognosis in cases of
organic heart disease is decidedly bad, both as to recurrence of the
chorea and as to the life of the patient, since with each succeeding

attack of chorea of a severe nature the systemic failure is great. Such patients usually die of organic heart disease (endocarditis) within two to five years after the original attack of chorea. The distinctly atrophied condition of the mucous membranes in these cases is shown by the fact that portions of the mucous membrane of the nose will be discharged *en masse*. In the milder cases that recover even after a series of attacks, the patient usually grows into vigorous health, no condition of previous cardiac trouble remaining in after-life.

Treatment.—This depends upon the severity of the disease. The average case can be given ambulatory treatment. Physical and mental rest for a few hours each day is essential, as is quietude at all times, and fresh air and sunlight. The child should be kept away from exciting influences of all sorts, particularly school; even agreeable excitement is harmful.

The emunctories should be carefully guarded. Copious draughts of water between meals are indicated, especially in cases giving evidences of auto-intoxication. The diet should be simple, consisting largely of milk, given at stated intervals (3 to 6 ounces every three hours during the day); the patient can also be given whey, buttermilk, largely to act as a diuretic, junket, soft-boiled eggs, bread a day old, and the lighter meats, avoiding carbohydrates and hydrocarbons, which disturb digestion in these cases.

In bad cases the patient should be put to bed absolutely, and where the movements are violent, padding about the bed should be judiciously done to prevent injury to the patient. Restraint will sometimes have to be resorted to in these severe cases. Salt-water sponging either with sea-water, or that *artificially made* by placing half an ounce of common salt to a quart of water, at a temperature of 100° F., will be a soothing measure, when applied by sponging for five minutes twice daily. Light *effleurage* is a measure assisting in soothing the nervous system, as well as aiding the circulation and bettering the nutrition.

Among drugs, iron is indicated. It should be employed especially in the anæmic cases of chorea. In children the tincture of chloride of iron, from 10 to 30 minims, given in water, three times a day, is of great value. This acts as a stomachic as well as an hæmatic. The teeth should be carefully guarded from the acid, rinsing the mouth with a solution of *bicarbonate of soda*. The French pill of Blaud, consisting of equal parts of sulphate of iron

and potassium carbonate, is a valuable remedy. In my experience this pill acts better in the older cases. The treatment of the disease *per se* is principally alterative, and no remedy acts quite so well as arsenic, which in the form of Fowler's solution is pleasant to take; also the dose can be nicely graduated to the necessity of the case. The usual method of administration is by giving 3 drops in water after meals, gradually increasing each dose by one drop, as shown in the accompanying table:

	BREAKFAST.	DINNER.	SUPPER.
1st day	3 drops	3 drops	3 drops
2d "	3 "	4 "	3 "
3d "	4 "	4 "	3 "
4th "	4 "	4 "	4 "
5th "	4 "	5 "	4 "
6th "	5 "	5 "	4 "
7th "	5 "	5 "	5 "
8th "	5 "	6 "	5 "
9th "	6 "	6 "	5 "
10th "	6 "	6 "	6 "
	etc.	etc.	etc.

This can be increased to 15 or 25 drops. When the physiological limit is reached the dose should be reduced one half, and kept at this dosage during the active course of the disease, unless recurrence of the arsenical symptoms are noted. In some persistent excitable cases the use of the bromides is indicated; particularly the ammonium bromide should be used, since it is less depressing to the heart. This salt will be found of special benefit in chorea with gastric irritability. Quinine, antipyrine, cimicifuga, and sodium salicylate are of value in rheumatic cases.

The cardiac symptoms should be very carefully guarded in a given case. Tincture of strophanthus is an excellent remedy for deficient compensation, and should be given in 10- to 15-drop doses three times a day until circulation is restored to the normal. Where serious organic heart disease complicates, digitalis should be employed. Long rest should be enjoined after the chorea has been recovered from. Cod-liver oil is a valuable reconstructive agent to be employed after the acute attack of chorea has waned. It is often desirable to change the scene in mild cases of chorea. Sea air, where high winds do not prevail, is desirable. High altitudes—above 2,000 feet—are never good for the patient.

HABIT CHOREA OR HABIT SPASM

This is a disease of childhood, having its origin in a previous attack of simple chorea or developing primarily. Heredity plays an important part in the *ætiology*, as does simulation of another case. Children of robust physique, who have developed a simple chorea of very active type, or, indeed, in whom the chorea has been of mild form, are very apt to be those who will have a habit spasm remaining after the simple chorea is apparently cured. Other exciting causes of the development of primary habit chorea are reflex causes of many sources, such as eye-strain, nasal disease, binding or irritation of some part of the body by clothing, a phimosis, seat worms, deformities of speech mechanism, such as tongue-tie or enlarged tonsils, or anything interfering with the functionation of the unstable nervous system. Thus, a boy, with the predisposition, upon donning his first pair of " suspenders " may develop shoulder twitching; or the child with tongue-tie may begin a contortion of the neck, induced by the endeavour to speak properly; or blinking of the eyes may result from an uncorrected astigmatism.

Symptoms.—The parts most frequently affected in habit chorea are the eyelids, facial muscles, neck muscles, and shoulder-girdle muscles—parts particularly sensitive in their nerve supply. The nature of the movement is that of a spasmodic rather than true choreiform motion, usually excited by mental or physical endeavour, although the patient can frequently control the movements in part by the will. They are absent during sleep, but, unlike simple chorea, are worse when the attention is not drawn to the condition at all. Inhibition of the movements exists more decidedly than in simple chorea, in the latter of which any form of mental action exaggerates them. As inferred, the patient's physical vigour is generally excellent in cases of habit spasm. In some cases where it is markedly localized, as in the posterior cervical muscles, they may become actually hypertrophied from overuse in these violent twitchings. There may be speech defects, such as echolalia, where sounds heard are reproduced; coprolalia, or the sudden utterance of profanity, etc.

Prognosis.—The duration of the disease is indefinite, but a favourable prognosis can be given in proportion to the movements and the cases resulting from previous simple chorea. Where it develops from some other exciting cause, as peripheral

irritations, in my experience, the disorder is sometimes seen lasting for years or becoming incurable.

Pathology.—This is not known.   Irritability of the upper motor neuron probably exists from some unknown poison.

Diagnosis.—It may be confused with Sydenham's chorea.   Persistency of habit spasm distinct from typical choreiform movements, also the absence of rheumatic history, are important points. Habit spasm from hysterical chorea is distinguished mainly by stigmata existing in the latter disease.

Treatment.—This consists in getting rid of every source of irritation, such as by correcting eyes, by circumcision, extraction of misplaced tooth, removal of enlarged tonsils, or operation for tongue-tie, etc.   If the case is the resultant of simple chorea, rest cure and tonics are principally indicated.   Moral influence is of vital importance in the therapeusis.   Hypnotism is a means, which, if it can be induced in the subject, is a quick agent towards cure. A *valuable* measure is to have the child stand before the looking-glass for three to five minutes, making an effort to control himself, and in this way educate the nerve-centres to proper inhibition.   Among drugs, the use of bromides, sparingly, or hyoscyamus may be of value until the nerve-centres become less irritable. Proper resort to bathing is a valuable measure, and should be used.   The cold shower-bath at a temperature of 60°, given every morning, will do much towards restoring nervous control in these cases, largely through establishing better equalized circulation within the central nervous system.

HEREDITARY CHOREA

SYNONYMS: *Huntingdon's Disease or Chorea*

This is a disease beginning at about thirty years of age, more frequent in the male sex, and characterized by its chronicity and tendency to mental affection.   The hereditary taint of this malady is not always directly in the ancestry, but there is always some neural vulnerability or cause for degeneration, such as chronic alcoholism, syphilis, etc., in the immediate progenitors.   The patient in the onset will scarcely have been seen by the physician, but it will be reported to him that the disposition has been changing for some months, the patient having become nervous and irritable, forgetful and perverse, and at times showing violent temper.

The movements generally begin in the upper extremity, are larger in the gyrations than in Sydenham's chorea, the head and neck becoming soon affected, and with the movements of the lower extremities the gait is particularly characteristic. The patient in his endeavour to walk slides and pushes one limb forward, then attempts to draw the other in its wake; then the other limb will be advanced in the same fashion, thus making with all its irregularity a more or less rhythmical alternation by the hampered voluntary effort. The movements of the trunk become marked within two or three months. These may cause compression of the abdomen and thorax. Occasionally involvement of the diaphragm produces sudden explosive sounds, similar to echolalia of habit chorea. The mental deterioration will have been well manifest by the time the choreiform movements, etc., as described, begin. This now consists of periods of mental depression, alternating with maniacal tendency, with forgetfulness, and the mental status tending towards dementia, the patient using finally a sort of jargon in speaking, entirely distinct from the mechanical disturbances of speech which also exist, due to the involvement of the pharyngeal muscles. So that careful observation has to be indulged to determine in foretelling the rapidity of downward progress of the case. The tendency to suicide is a characteristic symptom of the mental derangement. The patient's movements finally become so exaggerated that he cannot walk about, the irregular choreiform movements continued during the waking state, which, however, entirely cease when the patient is asleep. The movements also obey those of simple chorea in regard to the effect of mental excitement, physical endeavour, etc. The movements lessen only as mental deterioration progresses. This in time amounts to an active paresis, the patient becoming bedridden and dying of some intercurrent malady.

Duration.—The patient may be a sufferer for six or eight years before death supervenes, although the majority of cases are carried away within four or five years after its active development.

Diagnosis.—The diagnosis of Huntingdon's chorea must be made between it and *simple chorea*, or *habit chorea*, or *hysterical chorea*. The age of the patient, the recovery and relapses, and history of rheumatism and milder grade of movements would distinguish the simple chorea. The localized spasmodic disorder also occurring in children without any mental disturbances would dis-

tinguish the habit cases. Hysterical chorea would be known by other evidences of hysteria, the vacillation of symptoms, the absence of dementia and suicidal intent, and the hysterical chorea being more frequent in females.

Prognosis.—This is absolutely bad, no cases having been in any way permanently benefited.

Pathology.—This consists of congestion of blood-vessels in the cortex of the brain and final resultant irritability and degeneration of the central motor neurones, with areas of softening in those cases where the symptoms and signs of paresis develop.

Treatment.—The treatment of the disease is, as far as known, therefore, entirely of symptomatic nature. Supportive measures is the first desideratum. A simple nutritious diet, consisting of proteids in part, such as red meats, oysters, milk and eggs. Excess of carbohydrates should be avoided in all instances, and particularly in those where there is a diathetic tendency, such as to rheumatism or rickets. The system should be kept well flushed with water to prevent the retention of autochthonous and other forms of poisons, particularly absorption of ptomaines from the intestinal tract. The use of iodide of potassium or salicylate of soda, in small doses, is indicated where uric-acid retention is suspected. Such nutrients as cod-liver oil and hypophosphites and the lime-salts are valuable adjuvants. A pill of phosphorus, $\frac{1}{100}$ of a grain, or extract of chirata, 1 grain, thrice daily, are of service. Tincture of hyoscyamus or tincture of conium are antispasmodic medicaments that are sometimes of value in controlling the movements. The careful use of bromides to overcome the restlessness and general irritability is desirable. Hypodermic injections of hyoscine hydrobromate, $\frac{1}{100}$ of a grain, increased to $\frac{1}{50}$, acts markedly well in cases with maniacal disposition. Patients should be constantly guarded as to the possibility of committing suicide, and if paresis becomes a prominent and permanent symptom, it will become essential to commit the patient to a hospital for the insane.

CONVULSIVE TIC

Synonyms: *Tremor, or Giles de la Tourette's Disease*

This is an affection characterized by large irregular choreiform movements, involving the face and neck, at times affecting other parts of the body, and which may become general. It is also char-

acterized by large movements, and further by peculiar mental disorder. Heredity is a strong predisposing factor in this disease. Fright or shock is the *most frequent exciting* cause. By some authorities it is designated a psychosis, similar to hysteria. It usually begins in early childhood, and is accompanied by three prominent sets of manifestations. First, the involuntary movements, given in the definition—these being extreme contortions of the part rather than any typical form of movement. The face is thrown into a most grotesque physiognomy. The head is rotated and moved about irregularly upon the shoulders. In semiclonic spasm the movements extend first to the shoulders, causing a spasmodic shrugging, depression, and throwing back of the same. The trunk is finally involved in the spasm, and at length the extremities may be the seat of the wildest movements. All of these are exaggerated by excitement or voluntary muscular effort. At times the spasm is so great that the patient may be thrown to the ground. Peculiar involuntary sounds are emitted, due to the spasm of the glottis and pharyngeal muscles. These echolalia or coprolalia may be manifested in sudden exclamations. The mental disturbance consists of morbid fears, of doubt (misophobia), of disease (pathophobia), etc. Morbid impulses such as erotomania exist. Imperative movements may be attempted, but are usually frustrated by the involuntary retraction of muscles. The patient may finally become extremely delusional and pass into chronic terminal dementia, accompanied by wasting of all the muscles. Death from exhaustion will then supervene.

Diagnosis should be made from hysterical affections without difficulty, and would be clarified upon noting the persistent downward course of convulsive tic without the remissions of hysteria.

Prognosis is absolutely bad, there being no instances of betterment through any forms of treatment. The malady is an acute form of degeneration of the nervous system, a diffuse lesion being found in the central neurones, although of no definite distribution in any particular part.

SALTATORY SPASM

Synonym: *Jumper's Disease*

This is a neurosis allied to hysteria, occurring in early adult life in endemic form, often due to the simulation of other cases of the disease, and characterized by sudden violent spasms of the

flexor muscles of the thighs and calves in particular, which, in contraction, produce the typical symptoms of the disorder—a series of skips or jumps, keeping up for a few moments, and finally ending in enfeeblement or collapse of the patient, again to be renewed upon recovery. These remissions may occur many times during the twenty-four hours, the patient after each *séance* becoming utterly exhausted. Then a disturbed sleep will follow the attack. But the "jumping" begins on waking. The duration of the attack or of the affection is indefinite. Both are pretty much in proportion to proper treatment instituted. Such cases quite frequently develop during religious revivals, and will disappear after the excitement is over. Some may die of exhaustion.

Pathology.—There is nothing known of the pathology. Given an emotional subject, such irritability of the motor neurones may be set up by excitement; the only clue we have to pathogenesis.

Diagnosis.—This is made clear by observing the endemic form of the disease, the peculiar paroxysms, and the hysterical mental aspect.

Prognosis.—This is good. Most cases should recover.

Treatment.—Cure depends upon the ability to gain control of the patient and to impress his moral and mental nature. Hypnotism is a valuable therapeutic measure in this disorder, for, if the patient's mind can be concentrated enough to subject him to hypnosis, the case can generally be turned towards a rapid cure. The separation of the patient from the predisposing cause, or from others affected with the same malady, is, of course, to be insisted upon. Such conditions are allied to the hysteria observed in the northern coal regions of Pennsylvania four or five years ago, as investigated by Professor Newbold, of the University of Pennsylvania.

THOMSEN'S DISEASE

SYNONYMS: *Paramyoclonus multiplex; Myotonia congenita; Myospasm or Convulsive tremor.*

It is a congenital and inherited disease of the muscles, which is manifested by irregular bilateral spasmodic contractions of the muscles, 5 to 120 a minute, either in groups of muscles in the extremities, or involving all the muscles of the body. The disease appears earlier in life than the preceding, starting in early child-

hood, is much more frequent in the male sex, and is of hereditary nature. The patient while sitting will suddenly develop violent spasms of a part which may be sufficient to dislodge him from the sitting posture, or, if in progression, may cause him to tumble about or fall. The muscular contractions are accompanied by slight pain, but there is no tenderness over the muscles, and during the time of contraction they stand out in great prominence. The movements are generally quieted by rest, and are altogether absent during sleep, although the spasms themselves prevent somnolence in many cases. There is not the mental deterioration that is seen in convulsive tic.

Exercise or catching cold aggravates the spasm.

Pathology.—Little is understood of pathogenesis, and save for the hypertrophy of the muscle fibres and the constant proliferation of the muscle nuclei, nothing is definitely known of the pathology. The duration may be ten or fifteen years, the patient finally succumbing to intercurrent disease.

Diagnosis.—It cannot be mistaken for other maladies on careful study of the above outlined semeiology.

Treatment.—Massage, Swedish movements, and hot-water bathing are methods of relaxing the muscles. Nutritive measures, the hypophosphites among drugs being of value in keeping up the tone of the nervous system. Protonuclein may be of service through supplying nuclein to the degenerating muscle cell (gr. v, t. i. d.).

EPILEPSY

Definition and Ætiology.—Epilepsy is a neuro-psychosis in which unconscious attacks and a tendency to convulsive seizures are permanent features of the disorder. The disease is well known to be hereditary as to the degenerative *predisposition*; and it will frequently be found that where epilepsy has not existed in the previous generation there will at least be a history of alcoholism or specific disease in the ancestry. The disease may occur at any period of life. In those more truly hereditary cases (75 per cent) it begins quite early, before the tenth year. The sexes are about equally affected. It can be pretty positively stated, the more advanced the age at the development of the disease in the so-called *idiopathic* case, the less likelihood of heredity being an ætiological factor; but up to the degenerative period of life, when inhibition

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is less stable, epilepsy *may* appear as real stigmata of a degenera-
tive tendency.   Other causes of epilepsy are mental shock, injury
to the head, such as fractures, concussion or compression of the
brain, meningitis, brain tumour, gunshot wounds, toxæmias (alco-
hol, lead, diabetes, uræmia), reflex irritation (eye-strain, phimo-
sis, nasal or intestinal disease), and circulatory disturbances.   Cor-
tical apoplexy or meningeal hæmorrhage is not infrequently the
fundamental cause of the epilepsy coming on, due to insuperable
obstacles at birth; and in such cases the epileptic disease is but a
symptom of the cerebral palsy of childhood.   This is well marked
in the case of a boy under the writer's care who is spastic, has a
dolichocephalic head, has had convulsions since babyhood, and has
from three to ten attacks of *grand mal* daily.   He frequently suffers
from post-epileptic palsy.   Hydrocephalus is a cause of epilepsy.

Diagnosis and Symptoms.—Epilepsy is divided into major
(*grand mal*) and minor attacks (*petit mal*).

Major epilepsy is divided into *focal* or Jacksonian, where the
spasm is limited to a part or member of the body, or where the
convulsive seizure begins locally.   There are also *general* epileptic
convulsions.   Where the disease is well established there is present
a peculiar physiognomy, one of mental depression and instability of
purpose; also acne on the face, as a rule, the result of prolonged
use of bromides—the so-called *epileptic facies* when associated
with the features of mental instability mentioned.

Premonitory symptoms frequently precede the attack.   These
consist of headache, giddiness, jerkings, mental depression or ab-
erration, etc., and may occur for some hours before the attack.

The *attack* may be ushered in by a peculiar subjective sensation
called an *aura*; usually a *globus* or a feeling of depression, as of a
cloud coming over the patient, the vision becoming dull, and in a
moment passing into the convulsion.   There are many auræ.

Sensory Auræ.—The aura may also be a pain in the epigas-
trium—gastric aura; or rarely, it may be of paræsthesia in an
extremity; or less frequently, an hallucination of smell or taste
or hearing or sight.   I have known a case where the odour of
violets preceded the attack; another in which the sound of the dis-
charge of a gun was the hallucination; while irregular scotoma
or the seeing of some grotesque object may be the visual disturb-
ance preceding the seizure.   *Motor* auræ as a marked tremor may
occur.   Following this there may or may not be irregular auto-

matic movements in different parts of the body, the patient quickly becoming pale, then cyanotic and unconscious. In the light attack (*petit mal*) the patient may, with or without this precedent phenomena, pass into unconsciousness, accompanied by dilatation of pupils, and if at table, dropping the fork or some other object —the spell lasting but a few seconds, when the patient may resume his usual condition. The attack may be succeeded by somnolence or sound sleep lasting a half hour or more. In case the attack is one of *grand mal* succeeding the *premonitory signs* already mentioned, or with the *initial cry* in most cases, due to the firm rigidity of muscles of the chest, the patient falls over unconscious and goes into a general convulsive seizure, consisting of *tonic* rigidity followed by *clonic* spasms accompanied by frothing at the mouth and *conjugate deviation* of the eyes up and out towards the side of most violent movements; also dilated pupils. Blood may also be seen coming from the mouth, due to biting the tongue in the attack. The convulsion finally ends by rigidity of the flat trunkal muscles, and within a minute or so the entire attack is completed, there being rather sudden restoration of consciousness and of power in the extremities, although the intellect remains especially dulled for some moments, and there may be transient aphasia. The patient here again is apt to go into somnolence, which may last half an hour, or several hours in rare cases; but exceptionally *post-epileptic* paralysis may occur or *automatic acts,* the patient going about without mental control or appreciation of his surroundings. In an attack the patient usually suffers vesical or rectal incontinence, or both.

Occasionally "masked epilepsy" or *substitution* phenomena take place of the attack *per se,* the patient passing through automatic acts instead of a convulsion; or a condition of *double consciousness* may exist in which the patient may wander off for some hours or days, "coming to himself" in a remote locality. This is termed *ambulatory* epilepsy. Rarely, too, some marked psychosis may be the *substitution*, as a mania or delusional insanity with homicidal or suicidal tendencies. If the attacks follow one upon the other in rapid succession, we have what is termed *status epilepticus,* and in such condition the patient may actually die of exhaustion.

The epileptic complains of soreness and pain in the limbs *after* a seizure, and the body may be bruised or dislocations occur from the movements or in the falling at the beginning of the attack.

Convulsions occur more frequently in the early morning hours when the patient is asleep; this is probably due to the lowered physiological ebb of the nervous system at this time. In chronic cases this rule will not be maintained; the patient will have the attack at any part of the day (*diurnal*) or hour of the night. If it be *nocturnal* epilepsy, frequently the patient is not aware of the *attack* except for the soreness in the muscle. Intercurrent diseases or pregnancy may stay attacks for the time being.

Gowers mentions the following as the chief characteristics of *petit mal*, arranged in the order of frequency:

1. Sudden momentary unconsciousness, or "fainting," or "sleepiness," without warning.
2. Giddiness.
3. Jerks or starts of the limbs, trunk, or head.
4. Visual sensations or loss.
5. Mental state: Sudden sense of fear, etc.
6. Unilateral peripheral sensation or spasm.
7. Epigastric sensation.
8. Sudden tremor.
9. Sensations in both hands.
10. Pain or other sensation in the head.
11. Choking sensation in the throat.
12. Sudden scream.
13. Olfactory sensation.
14. Cardiac sensation.
15. Sensation in nose or eyeball.
16. Sudden dyspnœa.
17. General "indescribable" sensations.

After these attacks patients may perform automatic actions, as after attacks of *grand mal*. Eating at times induces attacks reflexly.

Convulsions usually sooner or later develop, and the two forms often coexist and interchange in the nature of attacks. In either form of epilepsy there is excess of leucocytes and poikilocytes.

Diagnosis.—Epilepsy may be confused with the hysterical paroxysm in rare cases. In hysteria there are stigmata of the disease, such as reversed colour fields, hystogenic zones, etc.; and the attack itself is prolonged a half hour or more, the spasms being more irregular and tonic in character. The patient bites the lips rather than his tongue. The eyelids are kept closed.

The pupils are not dilated. Syncopal attacks may resemble *petit mal*. Careful study of the circulation will differentiate.

Puerperal convulsions are told by the history of the case, the finding of albumin in the urine, and the more prolonged series of convulsions. Other symptomatic epilepsies are easily diagnosticated. *Myoclonic* epilepsy is told by the preceding myoclonic state of the muscles.

Pathology.—This is most likely a subtle anatomic or physiological affection of the cerebral cortex in all cases, though careful search in the majority of so-called idiopathic cases fails to reveal any changes by our present methods of research, which only goes to show our profound ignorance of this enigmatic disease. If a patient dies in convulsion, venous engorgement and minute hæmorrhages may be found in the cortex. Diffuse sclerosis of the cortex has been found (tuberculous or otherwise), meningo-encephalitis. Féré has frequently found neuroglear proliferation, while Marenesco and Block have described vascular and perivascular changes in the cortex. Hypertrophy of neuroglear bundles between pia and cortex is very frequent. Organic epilepsy will have as its pathology the pathology of the causative disease.

Prognosis.—An axiom in prognosis of epilepsy is, that the younger the case the worse it is for help or cure; the older the person, *per contra*, the greater hope for amelioration or recovery. Among the latter class we have subdivisions: *first*, cases in which mental degeneration takes place rapidly; and *secondly*, those where the attacks do not seem to produce any great mental depreciation. Napoleon's case was an example of this class. Focal epilepsies are pretty positively due to some definite organic lesion. In proportion as the cerebral cortex itself is involved will surgical aid fail of relief. But where such lesion is *operable*, as, for example, a localized meningeal lesion or depressed fracture, operation at the earliest moment will be fraught with relief or cure in 15 per cent of cases. After five years surgery cannot help epilepsy, since the habit has been too deeply established. In passing, it should be stated that extradural hæmorrhage in the newly born ought to be treated surgically, if sequent epilepsy from a secondary irritation is to be avoided in later life. Such surgical procedure has as yet never been done, but has been suggested by Dr. S. Weir Mitchell and Dr. W. W. Keen. As refinements of diagnosis become more and more accurate in the new-born will the

possibility of such help from the surgeon be determined. Spontaneous cures rarely occur. If attacks have lasted two years, there is little hope of cure in " idiopathic " cases.

Treatment.—The above-indicated surgical treatment we most earnestly commend in early cases of the disease where there is any evidence of dural hæmorrhage, or other foreign body causing irritation of the cortex. A child with epilepsy should be subject to most rigid scrutiny for evidences of reflex irritations. Gastrointestinal disorders, including those due to parasites, as the nematodes or trematodes, should be eradicated by first expelling the offending parasite. The use of depresso-motors is indicated in epilepsy, since there is excitation of the motor cortex; such symptomatic treatment is, of course, to be desired in itself, and these drugs do much good in this disease. Ten to 15 grains of the bromide of potassium should be given three to four times a day, according to the age of the patient and the physiological idiosyncrasy. In giving the bromides, they should be continued for a month or two, when, even if the attacks are stayed, it is wise to withhold the drug for a week, then to continue with small doses, gradually getting up to the maximum, as mentioned. The opium treatment of epilepsy, which consists in the gradual increase of the drug up to producing stupor in the patient, has been commended by Collins in this country and by Flechsig and others abroad. The tendency to disturb the stomach of the patient or to produce the morphine habit seems to me a strong contra-indication against the use of the drug; although I have seen a hypodermic injection of morphine sulphate given during an epileptic status quiet the patient and prevent further seizures for the time being. The use of cannabis indica will be found of value in cases of *petit mal.* The berries of the horse-nettle or *Solanum carolinense,* especially in the form of the fluid extract, I have found beneficial in a number of cases of idiopathic epilepsy where there is no hereditary taint. A fluid extract of the drug is particularly serviceable if made from fresh berries, the drug being pushed to the full physiological limit. At present in a case under this form of treatment, after all other methods have failed, a fluid extract of the berries, pushed to a dram four times a day, has prevented an attack for three months, whereas the girl had been having them previously on an average of one attack every ninth day. Belladonna is occasionally a useful remedy, especially where the circulation seems to

have failed through vaso-contraction; this in conjunction with nitroglycerin, given in continued dosage, will sometimes act favourably in allaying the spells. The use of digitalis in combination with the bromides has controlled attacks in many cases with feeble heart circulation, where the bromides alone had failed. The reason of this lies perhaps in the better circulation produced in the brain, as well as in the more active circulation carrying off waste products. In all cases of epilepsy the gastro-intestinal tract should be particularly guarded. If indican be found in excess in the urine, especially is it important to use intestinal antiseptics, such as salol, grains 2 to 4, in twenty-four hours; also high flushings of the colon with lime-water solution, an ounce to a quart of tepid water. If there be gastric dilatation, lavage of the stomach will frequently, by preventing absorption of undigested food products into the blood, produce happy results. Regulated exercise is essential. The mind should be lightly employed. Special care should be given to massage and the hygienic condition of the skin; salt bathing is particularly desirable to stimulate cutaneous circulation, and to the full extent relieve any congestion of the meninges. Toulouse speaks of the value of withdrawing common salt from epileptics, thereby permitting free absorption of the bromides. Disturbed metabolism, especially the uric-acid diathesis, calls for careful attention; and in the latter class of cases the use of alkaline waters is to be commended. Milk diet, or at least the withholding of meats and indigestible foodstuffs, is important. The worst cases are better treated in the colony farms, as at Sonyea, N. Y.

NEURASTHENIA

SYNONYMS: *Nervous exhaustion; Nerve tire; Nervous prostration; Beard's disease.*

Definition.—This is a functional disease in which there is a condition of irritability with weakness of the central nervous system. It was first described by Beard, of New York, in 1869. The age of onset is usually between eighteen and thirty years. It is more frequent in women (75 per cent of the cases), more persistent when occurring in the male. The disease is *hereditary* or *acquired.*

Causes.—Overwork, worry, hereditary predisposition, poisons, autoinfections, and fright. Other predisposing causes are anæmia and sedentary habits, lack of exercise, unhygienic surround-

ings, absence of sunlight, improper food, and genital disease, particularly in women. (See Fig. 64.)

The communications of the nervous system to the genital organs may be better understood by the following diagram:
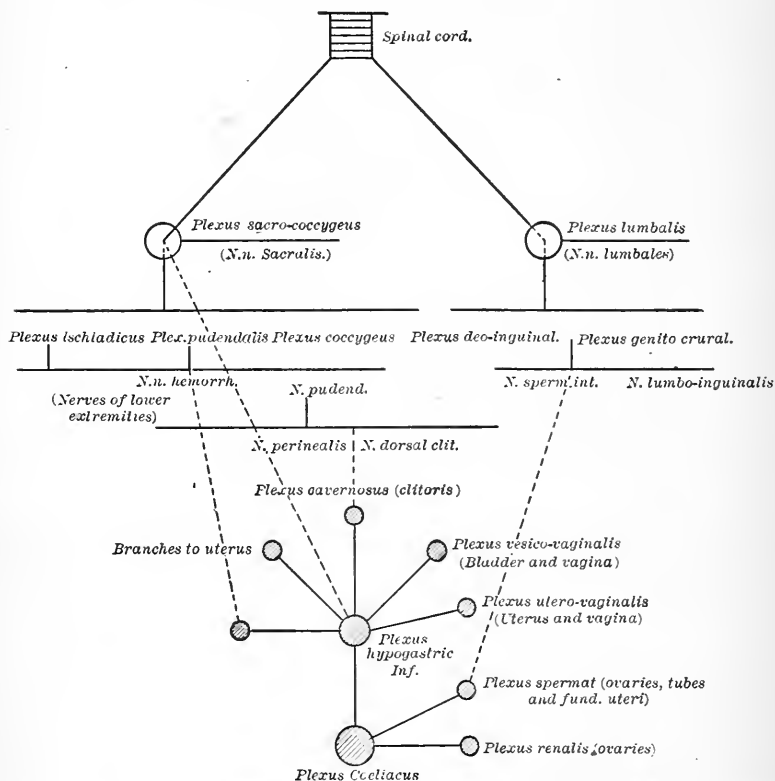
FIG. 64.—COMMUNICATIONS OF THE NERVOUS SYSTEM TO THE GENITAL ORGANS. Dotted lines indicate combinations. (Pearce-Beyea.)[1]

From this description it is definitely shown that there is an anatomical relation between the female genitals and the nervous system, and it would seem that there must be, at least in some measure, a communication between certain physiological functions and diseases of the female genitals and the nervous system; and also, therefore, to some diseases of the nervous system.

Symptoms—Psychic.—Generally an irritability of the mind, lack of power of concentration, forgetfulness and inability to carry on a continuous line of thought; peevishness, and supersensitive-

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[1] The Memphis Lancet, September, 1898.

ness, with insomnia. The patient complains of vertigo. Photophobia or other special sense irritability may be manifest. The bladder may be " irritable " and an early symptom.

Sensory.—Peculiar sensations exist as of heat and cold, paræsthesia of the extremities, or actual hyperæsthesia. There are usually tender spots on pressure over the spinal column, principally over the cervical, dorsal, and lumbar enlargements of the cord. The patient complains of a sense of constriction about the head, a vague feeling of distress, often described as a " helmet-like " headache. The pain may be localized particularly in the frontal and occipital regions. It is made worse by use of the eyes and by mental or physical work. Backache is constant.

Motor.—These consist of a fine tremor of the extremities, and tongue, and lips. The tremor is brought out by intention. It is apt to subside when the patient is quiet in the milder cases. The muscular tone is lessened, fibrillary contractions of the muscles may occur, especially if tapped with the percussion hammer, that may bring this phenomenon out enough to simulate the fibrillary contraction of degenerative states, as in chronic poliomyelitis. There is widespread enfeeblement of the musculature. The patient tires on the slightest exertion, but there is never palsy or convulsion. The reflexes are heightened. Weakness is worse before noon.

Vaso-motor palsy and leakiness of the skin with cold extremities is frequent. Palpitation of the heart is not uncommon. The urinary secretion is usually increased and of low specific gravity. with excess of phosphates. Indicanuria is very general in cases of gastro-intestinal origin. Colliquative diarrhœa is a common symptom, although it may alternate with persistent constipation. The patient often complains of tympanites, borborygmus, and tenesmus. Hyperchlorhydria is not uncommon, and is especially marked after a meal. In some cases there is distinct gastrectasia. Displacements of the abdominal organs will be found in a few cases of neurasthenia ; thus floating kidney is not a rare occurrence ; displacement downward of the liver we have seen, due entirely to the relaxation of its ligaments. The occurrence of abdominal pain in the right lower quadrant of the abdomen is undoubtedly due to a fermentation in the cæcum. and while some cases have been mistakenly diagnosed appendicitis. it is possible that neurasthenia may be responsible in this way for that disease in a few instances.

Pathology.—There has been found by Hodge granular change

of the cytoplasm of the neurone, following induced exhaustion, in bees. This is accompanied by a diminution in size of the cell, but without actual degeneration; the incipient pathologic state being recovered upon the restoration of the bodily function. In the human body it is likely that this same change occurs, which has, however, not been actually demonstrated as yet.

Adjunct pathogeneses of neurasthenia are circulation of the blood to the cerebral cortex, of toxines, alloxuric bodies, uric acid,[1] and allied by-products. The deficiency of excretion of solids by the kidneys in a few cases may be the exciting cause for the depression of the nerve-cell, and, therefore, induction of active symptoms of nerve exhaustion.

Diagnosis.—Neurasthenia is to be told from hysteria, in the latter of which the emotional state of the patient is the salient point. In the hysterical person, too, there is no muscle hypotonia, as in nerve exhaustion; perversions of common sensation, paralysis, or convulsions being frequent symptoms in hysteria. The inconsistent symptomatology is a vital point in determining hysteria. Asthenias following prolonged fevers are to be distinguished from neurasthenia *per se*, since in these symptomatic disorders the patient has not the particular irritable symptoms already mentioned.

Secondary neurasthenia is to be told from *primary* or essential neurasthenia by finding a reflex source of irritation for the former, such as uterine diseases, phimosis, or sexual disorder in the male (the so-called *sexual neurasthenia*). *Neurasthenia terminalis* is the chronic form that has become incurable.

Prognosis.—The prognosis of acute neurasthenia is good if treated at once, depending upon the non-hereditary tendency of the patient. Cases with the hereditary taint are to be given a guarded prognosis. Cases of secondary neurasthenia will also recover promptly in proportion to the relief of the primary exciting cause, such as a proper repair of cervical and perineal tear with prolapsus uteri; the correction of errors of refraction, etc.

The duration of the average case of simple neurasthenia is about three months. Chronic cases may last for years, with remissions of periods of several months, or longer.

Treatment.—Revolution in the treatment of nervous exhaus-

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[1] See Laboratory Studies on Uric Acid in Neurasthenia, etc., American Journal of Insanity, vol. lviii, No. 1, 1900.

tion was made by Weir Mitchell in 1874, when the different meth-
ods employed in treating this disease with more or less success
were correlated and established into guiding principles. These
consist of (1) mental and bodily rest, (2) full feeding, (3) isola-
tion, (4) massage, as well as movements, and (5) electricity. The
employment of this combination is designated the " rest cure." It
should be carried out for a period of six or eight weeks to three
months. If good results are not obtained at the end of three
months, experience teaches that it is useless to continue the treat-
ment. It is essential, in carrying out the method, to remove the
patient from home, or, if this cannot be, to a remote portion of
the house, away as far as possible from the atmosphere of home.
The patient is kept absolutely in bed and given two good nurses, the
room being kept at an even temperature, at an average of 68° F.
All reading is to be discontinued, and even the nurses are in-
structed to converse but little over pleasantries of the day, and
none as to the patient's vagarious complaints. The instructions,
which should be explicitly written by the physician, are further
as follows: Patient put on a light milk diet, a glass being given
at seven o'clock in the morning, on waking, and then every two
hours during the day; thus from 9 to 10 doses of milk will be
given the patient in twenty-four hours. Beginning at first
with 3 ounces, finally reaching 8 to 10 ounces, which would make
27 to 72 ounces taken within the twenty-four hours. At any
time the patient's stomach rebels the increasing dosage of milk is
held or lessened, or lime-water or peptonization may be employed
to assist digestion. In addition to this dietary the patient may
take at 7.30 o'clock in the morning a cup of cocoa, which can be
repeated at eight o'clock at night. If there is persistent insomnia,
and the stomach is not being taxed for digestion too severely, a
piece of dry toast can be given in the evening with the glass of
milk to vary the monotony, although it will be found, in the aver-
age cases suitable for this treatment, that large quantities of
milk can be taken without causing any disturbance. The milk
diet is continued for three or four weeks, and when the patient
is gaining in weight the diet can be increased to solids, such as
proteids, in the shape of red meats and oysters, lamb chops, eggs,
etc. At this stage it is also well to add a nutrient, as the fluid
extract of malt, of which there are several varieties on the market,
the Johann Hoff malt being one of these.

As regards rest, the patient is not even allowed to sit up,

except for the moment of taking the food, which should be administered by the nurse. Nor is the patient permitted to read.

The patient should be given a tepid sponge bath, followed by an alcohol rub in the morning, and an alcohol rub alone at 9 P. M.

The braiding of the hair and general toilet should be left entirely to the nurse. These little details are essential in the proper carrying out of the *régime*, if good results are to be obtained.

The massage should be given at about 10 A. M., beginning at first with light *effleurage*, finally reaching the deep kneading of the muscles, which is of most value in their nutrition. This also assists digestion of so large quantities of food. It is interesting to note the relation of food supply to the proper massage and Swedish movements in a rapidly progressing cure under the rest treatment. Electricity usually is best in the form of faradism, the nurse employing it at about three o'clock in the afternoon, as follows: The extremities are first given the rapidly interrupted current, then the slow interrupted current is applied to the muscles of the upper extremities, lower extremities, abdomen, pectoral region, erector spine, and back group of muscles. (See *Massage* and *Electricity*.)

When the patient has gained in weight and in general nervous health she is gradually got out of bed, which should first be by sitting up in bed for five minutes at a time, gradually increasing this to twice a day, and finally the patient is to be lifted upon a chair. The length of time is increased until sitting up two or three hours, morning and evening, has been indulged. The patient can move about the room and then get back gradually to normal life—a renewed person. As a rule, improvement continues for many weeks after the case has been so treated.

The so-called *partial-rest* treatment consists in having the patient rest for an hour or so in midday, plus the hour's rest after the massage in the morning, the rest of the day being devoted to quiet living, when the patient may do a certain measure of work, if not of a taxing nature. The diet in partial-rest cure is never limited to milk alone, and malt is given with the meals. Frequently the use of phosphorus in pill form, $\frac{1}{100}$ of a grain and a capsule; of extract of chirata, 1 grain; extract of ignatia, $\frac{1}{4}$ grain, to be given after meals, will be of value. The simple elixir of iron, quinine, and strychnine is useful in the cases among the poorer classes, and reaches many of the indications.[1] In any case of neurasthenia the bowels should be kept

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[1] See Appendix.

soluble. For this purpose the Lady Webster pill or the pill of aloin (gr. $\frac{1}{8}$), belladonna (gr. $\frac{1}{12}$), and strychnine ($\frac{1}{60}$) may act nicely when given at bedtime. The fluid extract of cascara, in from 15- to 30-drop doses, is a valuable remedy in these cases. Where there is acidity of the stomach the phosphate of soda, in dram .doses, on waking, will prepare the stomach for better digestion. In this case the pill of nitrate of silver (gr. $\frac{1}{8}$), given three times a day, will be of service as a tonic to the mucous membrane of the stomach.

Hydrotherapeutics in neurasthenia is very important, and should be used as indicated, by sponging in rest-cure cases or applying cold douche in ambulatory cases. The Charcot's douche is one of the most valuable methods of applying water, and consists in the circular spray applied to the trunk, chiefly over the spinal column. In hydrotherapeutic procedures for neurasthenia it is best to begin with a tepid sponging or a few moments' bath of the body accompanied by frictions, and at the end of five or ten minutes follow this up by a cool spray, at a temperature of 65° F. This entire procedure is to be followed by a brisk rub with a Turkish towel and rest for half an hour afterward. Flushings of the colon with tepid water every other day is valuable in those cases of an autotoxic nature. The use of warm mud-baths, such as the " fango " of the Mediterranean, I have seen do good in cases accompanied by the most persistent insomnia. Where there is an irregular excretion of uric acid, especially in rheumatic diatheses, the use of salicylate of soda will be found of value in relieving the general irritability of the patient and painful headaches, probably due to by-products circulating in the meninges. The correction of the eyes should be had in cases of reflex ocular defects. All sources of irritation should be eradicated, as phimosis, etc. The gynæcologist is here closely related to the neurologist, and while too many cases of neurasthenia are assigned as gynæcologic, it must be stated that judicious study of neurasthenia, following childbirth, will determine it to be caused or aggravated by some lesion of the pelvic tract. Other measures to be employed in neurasthenia are fresh air, sunshine, and salt breezes. Iron should be employed in anæmic cases. Good food must be had at all times. Hypernutrition is an essential in the treatment of this disease. The patient should be encouraged at all times.

The climatology of neurasthenia is not sufficiently worked out

at the present time. It would seem axiomatic at all events that the patient should not ascend to an altitude above 2,000 feet. A very stimulating climate should be avoided, as should also districts menaced by fogs, cloudy, saturated atmospheres, with but slight movements of air currents, and the opposite, high-wind localities. Sea level, with continuous heat, non-varying, is baneful. Thus the Bermuda Islands and Florida are enervating localities. Ideal conditions for the neurasthenic patient include sea-air in a well-wooded country, far enough from the coast to avoid its fogs. Sea voyage is, as a rule, an excellent preliminary to other climatic measures, and, provided it is not stormy, will act both psychically and physically in soothing the nervous system.

HYSTERIA

This is a functional disease of the nervous system, manifested by psychic (emotional), motor, and sensory phenomena.

This disease has been the cause of much discussion as to its entity, and to many other misunderstandings in the progress of medicine than any other malady. It is the old " possessions " of the middle ages; in common-sense garb to-day a disease of emotions.

Ætiology.—Heredity is distinctly a cause of hysteria. Next in order would come moral shock, physical shock, and overwork, and sudden drain upon the system, as in acute anæmia, typhoid, etc.

Symptoms—Psycho-sensory.—These are predominating, being somewhat masked in certain cases, and therefore misleading in the differential diagnosis. There are two types of hysteria: the general state associated with *stigmata,* and the *paroxysmal.*

The following table from Gowers illustrates the differences between epileptic and hysterical paroxysms:

	EPILEPSY.	HYSTERIA.
Apparent cause,	None.	Emotion.
Warning,	Any, but especially unilateral or epigastric auræ.	Palpitation. malaise. choking, bilateral foot auræ.
Onset,	Always sudden.	Often gradual.
Scream,	At onset.	During course.
Convulsion,	Rigidity, followed by jerking, rarely rigidity alone.	Rigidity, or "struggling," throwing about of limbs or head, arching of the back.

	EPILEPSY.	HYSTERIA.
Biting,	Tongue.	Lips, hands, other people or things.
Micturition,	Frequent.	Never.
Defecation,	Occasional.	Never.
Talking,	Never.	Frequent.
Duration,	A *few minutes.*	More than *ten minutes,* often longer.
Restraint necessary,	To prevent accident.	To control violence.
Termination,	Spontaneous.	Spontaneous or induced (water, etc.).

Among stigmata of hysteria there is the emotional element of the patient, the lack of control of will power, which is induced the more by any slight excitement, particularly of a psychic nature. Narrowing of the visual fields and reversal of the colour fields (red being perceived before blue) is frequently found in hysteria. Hysterical amblyopia or hysterical blindness is a very rare symptom. The patient is able to escape objects in walking about, but has no conception of their shape, size, or use. There may be photophobia, achromatopsia, or dyschromatopsia.

Other psychic special sense symptoms are the following: Hyperosmia, anosmia, and parosmia; in the ear we have similar conditions—deafness, increased acuity of sense of hearing, tinnitus aurium; the sense of taste may be affected—hypergeusia, ageusia, and parageusia. Hysterical hyperpyrexia may occur.

The so-called hysterical zones are frequently found on pressure over the dorsal, cervical, and lumbar regions of the spine, also over the ovaries and beneath the breasts—*hysterogenous zones.*

Other sensory disturbances may be geographical or segmental or glove-like areas of anæsthesia, or a very sharply defined hemianæsthesia. This is quite distinct from organic hemianæsthesia, where the loss of sensation passes beyond the median line—due to interdigitation of nerve-fibres from each side upon the skin beyond the median line. *Hysterical* joint is a rigid, tender joint.

The subjective sensation of tingling in the extremities (paræsthesia) is very usual in hysteria. At times this takes on the hallucinatory expression of ants creeping over the body, and it is very difficult to convince the patient that this is not so.

Motor.[1]—The motor symptoms of the continuing state of hysteria are, as a rule, of excessive muscular action, with irregular

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[1] Hysterical ataxia may occur. Astasia-abasia is inability to stand or sit well in hysteria.

and rapid movements of the extremities in any particular voca-
tion. The depressive motor states do exist, however, in the minor-
ity of cases, where the patient is apathetic, inactive, or even seden-
tary, with or without actual palsy. *Catalepsy* or trance may per-
sist for days. Palpitation of the heart, rapid breathing, excessive
or deficient secretion of urine, or even hæmaturia, may exist in
hysteria. Dysmenorrhœa and amenorrhœa are not uncommon. I
have not observed any particular alteration of the function of the
bowels in hysteria, although regurgitation of food or even merycis-
mus and extreme tympanitis (*phantom tumor*) are very usual.

The *paroxysmal stage* of hysteria consists particularly of vio-
lent motor manifestations, which, as a whole, is described as the
attack of hysteria. In this the patient will, after some hours of
premonition, of which he will give ample warning, pass into local
and general convulsions, which will assume the tonic type in par-
ticular, the patient in her contortions being drawn into opisthot-
onus or pleurothotonus. The movements of the extremities are
more or less vermicular, rather than of true clonic or tonic type.
The eyes are rolled about, although the lids are generally kept
closed, and it will be found that the pupils are not dilated, as in
epilepsy. The patient will not injure herself as a rule, and falls
in the attack upon some convenient spot. While there may be
frothing at the mouth, there is never biting of the tongue, nor the
characteristic cry seen in epilepsy. The attack is noted for its
dramatic effect, and continues the more when the patient is in
special solicitude by her friends. An " attack " of hysteria may
last from five to ten minutes to several hours or longer, the
patient usually passing out of the condition suddenly, and may
from sheer exhaustion fall into sleep or a state of trance lasting
some hours. The patient is not unconscious in the attack, and if
pricked with a pin or otherwise irritated will frequently resist
and give evidence of the disturbance.

Certain passionate attitudes are described by the French in the
" grand " attack; these are seldom found in America. They are:
  1. Epileptoid.
  2. Grand movements.
  3. Period of passionate attitudes.
  4. Period of delirium.
The attack may last twenty minutes or as many hours.
Diagnosis should not be confounded with other diseases, ex-

cepting possibly with neurasthenia, where the emotional element
is wanting; but it must be remembered that the two diseases coex-
ist. Hysterical manifestations occurring as symptoms of brain
tumour should be borne in mind, and careful search made in all
cases for the symptoms in the latter disease. Hysteria is *very* apt
to be diagnosed in some cases of tumour of the frontal lobe, and
in this class of cases the most mistakes are made.

Prognosis is always good as to the paroxysm, and to be cau-
tiously given in cases hereditary in type. In acquired hysteria,
where the initial cause has been eradicated and impression has
not been made too profound upon the nervous system, recovery
will frequently occur after proper treatment.

Treatment.—This consists of psychic influence largely. Such
patients should be got entirely under the control of a physician,
away from sympathetic friends, and firmly, though gently,
treated. Encouragement should be given when the patient con-
trols herself, thereby strengthening the will power, which is so
deficient in this disease. If the system is run down, a tonic line of
treatment must be enjoined. Change of scene is frequently neces-
sary, a long sea voyage being desirable where there has been a great
moral shock as the exciting cause.

Among drugs bromides are indicated; asafœtida, gr. v, in pill
form; or a compound sumbul pill of Goodell, ext. sumbul, gr. j;
ferri. sulph. exsic. gr. j; asafœtida, gr. ij; acid. arsenicum, gr. $\frac{1}{40}$,
given thrice daily. In the attack the administration of aromatic
spirits of ammonia, valerian, or cold douches to the face will be of
value. Hypodermic injections of plain water, or where the con-
vulsion is serious small doses of morphine, guarded by atropine,
will be indicated; or a cold plunge may be given.

Counter-irritation to the spine, such as by the actual cautery
or blisters, will frequently give prompt relief to the paroxysm.

Hypnotism is a valuable means for the cure of certain classes
of cases, particularly those with local palsies, either motor or
sensory. It is of less value in cases involving the entire mus-
culature. Patients recovering from hysteria should be placed
in an entirely different surrounding, if the cure is to be permanent.
The care of the hysterical patient demands a keen insight into
human nature. It is in this disease that the doctor and nurse,
through judicious sympathy, alternating with rigidity of disci-
pline, that the " paralyzed will power " (for such is not an inapt

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FIG. 65.—HYSTERICAL PARALYSIS. BRACES AND WHEEL-CRUTCH APPLIED TO AS-
SIST IN WALKING TILL SHE GAINED POWER.—CURED.

description of the psychological state) greatly assists earlier and complete recovery of the patient. It is essential in managing this disease to know the history, the heredity of the affection or not, and then to learn the predisposing and exciting causes which have led up to the "attack," be it a passing syncope, or convulsion, or other form of the acute malady. It is these cases where especially, as some author has well said, the nurse should have an eagle's eye, a lion's heart, and a lady's hand in dealing with the case, one of those optimistic Greek maxims which gives vitality to all action. In treating any form of hysteria, we will find that 90 per cent of the cases cannot be aroused to sudden cures through such agents as hypnotism, mental suggestion, and the like; and while such measures should be practised in selected cases by the physician (with the nurse's aid), still a happy discretion in knowing when to abandon these more mysterious methods of cure, successful as they are at times, is to be impressed upon us. Therefore, in treating hysteria, it should ever be present in mind that these at times quicker forms of treatment cannot be continued when we have learned that there has been no rapid improvement in the symptoms. Local paralysis or spasms of a hysterical nature, are in my experience more easily controlled than generalized symptoms, by hypnotism; so that whether it be paralysis, motor or sensory, or of contractures, the subconscious state is much more difficult to influence by this means. In these cases the nurse, who is more constantly in attendance than the physician, by lending a spirit of hopefulness in pointing out to the patient from day to day improvements observed by the physician, will re-enforce therapeutics greatly. And our nurses should be better instructed in this fact that, when the lowest ebb of will power is established over months and years in extensive chronic paralytic forms of hysteria, nerve energy occasionally is never restored; and, therefore, function remains in abeyance without any pathological lesion taking place other than those accompanying the malnutritional state. The greatest exciting cause of hysteria is some form of fright, as is instanced in a case under the writer's care, which was awakened by a rat running across the bed, since which time she has had hysterical stigmata with generalized functional tremor. Another instance is that of a young woman [1] who fell down a flight of seven steps without any serious bodily harm, although a pain in the back led her to believe that she would

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[1] International Medical Magazine, September, 1901. (See Fig. 65.)

be paralyzed, which she finally was. She has now been bedridden for three years, with hysterical contractures, anæsthesia of the lower limbs up to the knees, and once a clonic hysterical spasm of the abdominal muscles; which latter was cured by producing the initial stage of ether anæsthesia. It took us many months before this woman could be impressed enough to show signs giving ground for hopeful prognosis, and later cure. I mention these rare cases so that the nurses may be taught to understand that nervousness is not all foolishness, and has some basis for its existence, perhaps, in the theory of immobility of the neurones; and therefore the diverting of the nerve-currents irregularly, which is very likely the fact, as suggested by Duval and Lugaro.

Nerve energy is, to my mind, but the most refined form of electricity. There are some physiological phenomena which tend to prove this, as in the sense of light-flash perceived when the oculist severs the optic nerve in enucleation, or in the sudden restoration of power and sensation following nerve suture, as in a case of traumatic operation of the ulnar nerve recently seen by the writer. We have in this theory a reason for the efficacy of electricity in nervous diseases.

TRAUMATIC HYSTERIA

Synonyms: *Railway spine; Traumatic back; Traumatic neurasthenia*

Definition and Causes.—This is a form of hysteria, the result of psychic and physical injury. By some it is thought to be a functional disorder; by other authorities to have an organic basis, the milder cases not having, as yet, the pathology fully determined. Others describe the condition as entirely mental. Unfortunately, this is a disease in which the wildest variation of opinion exists as to its nature. We shall give what is accepted by the best authorities. Traumatic hysteria is more frequent in neuropathic persons, is precipitated by some form of physical injury, in contradistinction to the idiopathic hysteria, so called, which usually has its exciting cause in some psychic trauma alone. Traumatic hysteria is more common in the male sex, in whom it is more severe, as a rule, than in the female sex. The reason for the more frequent occurrence in males is undoubtedly due to the liability to injury in men.

Symptoms and Divisions.—The history of its onset is usually

as follows: The patient is struck upon the back or thrown from a railway carriage, being discovered in a more or less condition of shock, with or without consciousness; and when he is restored the physical injury has impressed itself so upon him, that with the mental instability induced, the patient imagines that he is seriously injured, whether this be actually the case or not. On this latter depends the dividing course in the symptomatology. If the physical injury has been of slight nature the neurotic person will develop symptoms entirely psychical. Or, if an injury to the muscles or ligaments, which in time is healed, occurs, the length of time of the actual trouble will be prolonged in such a case by the fixed hysteria established during the course of the real trouble.

The second class of cases develop actual disease of the membranes and spinal cord of a subacute inflammatory character; or it may consist of hæmorrhage within the cord, or to irritable weakness (neurasthenia), and finally to degeneration.

The *first class* of cases presents the typical hysterical symptoms, plus some local manifestations, the result or supposed result of the injury. In the first instance, of course, the actual lesion is soon dissipated; in the second subdivision, the history of an accident plus these persisting localizing phenomena make plain the organic nature of the disease.

In the purely hysterical cases the patient usually complains of *rigidity* of the back, which upon physical examination is determined to exist as the result of tonic spasm of the erector spinæ groups of muscles. Along the spine there are points of exquisite tenderness apt to be localized over cervical, dorsal, and lumbar enlargements of the cord. This tenderness upon superficial pressure is more than upon deep pressure, to which there would be exception only in the cases where muscle or tendon injury still prevailed. In the latter case so-called "tender-spots" will remain after the muscle is healed at the site of the original injury. The patient becomes irritable, tires at the slightest exertion, mental or physical. Palpitation of the heart, bradycardia or tachycardia, may exist, and these symptoms frequently are not synchronous with the pulse-beat. *Per contra*, the pulse may be running, feeble, and irregular, while the cardiac muscle is not especially disturbed in function. This cardio-vascular condition gives evidences of the widespread irritability of the sympathetic nervous system, which is probably the more affected because of the relation of the gan-

glia to the spinal column, where concussion or a blow upon the back would transfer the physical effect immediately to these ganglia lying upon the bodies of the vertebræ.   Other *vaso-motor* changes are local or general flushing and sweating or coldness of the extremities.   Seldom is there a true cyanosis, however.   Other organs affected largely through the sympathetic nervous system are the stomach, producing gastrectasia, borborygmus, hyperacidity from fermentation of food, and intestines, causing alternating serous diarrhœa, abdominal cramp, and physical signs of pseudo-appendicitis.   The kidney secretion may also be affected, so that anuria, polyuria, or even dysuria may occur.   Irregularity of the sphincter muscle of the bladder may exist, inducing spasm or relaxation with symptoms of tenesmus, incontinence of urine, etc.

The knee-jerks and other deep reflexes are greatly increased, but the valuable point in diagnosis of this first division is that true ankle clonus is not present.   The muscles are flabby and do not show reaction of degeneration nor localized wasting.

Seldom do sensory symptoms occur other than paræsthesia or hyperæsthesia ; anæsthesia, as in simple hysteria, never occurring.

The psychic symptoms are sometimes bizarre, being entirely of hysterical nature, including stigmata and paroxysms of this disease in quite exaggerated form.   (See Hysteria.)

The symptoms of the *second* class of cases are again, in the majority of instances, largely of psychic nature.   In addition there are signs of organic disease.

The milder cases will present the signs of rigidity of the spine, already described, plus physical evidences of chronic injury to the tissues around, between, or within the spinal vertebræ.   The points of exquisite tenderness, made worse by the slightest movement, and perhaps with some superficial swelling, would indicate a myositis.   Deeper lying points of tenderness, also exaggerated by slight movement of the spine, with in some cases undue separation of the spinous processes, would indicate relaxation or tear of intervertical ligaments or disks, in the latter of which great pain would be experienced, when the patient should be assisted to stand on his toes and then dropped cautiously upon the heels.   These patients *are never in any degree of comfort,* are usually harassed by severe pain, though periodic and worse at night, preventing sleep.   Symptoms induced from the persistent insomnia may end in a mild grade of mania or even of delusions as to their friends,

especially where the doctor and relatives give out that there is no organic trouble present. The reflexes are greatly heightened, the musculature is frequently in hypertonia, due to irritation, and pseudo ankle clonus may exist. In the severer cases in this second class the membranes of the cord are congested or in subacute inflammation, which may in time extend to the spinal cord itself, producing passive congestion or low-grade myelitis, which can be determined by the discovery of persistent ankle clonus, involvement of the bladder, either incontinence or resultant cystitis, and rectal incontinence similar to an ordinary myelitis. A typical case of this sort the writer has recently seen where hysteria has been pronounced, and yet where, in spite of a large recovery in a suit for damages, the patient still has the evidence of organic disease referred to.

Diagnosis.—Simple hysteria is distinguished by the absence of a back injury. Malingering is told by the fact that the malingerer cannot consistently simulate the symptoms constantly.

Prognosis.—The duration of traumatic hysteria is very indefinite, the hysterical cases at times being almost as persistent and chronic as in organic change of the cord. Where organic lesions exist we cannot hope for entire recovery. The pure case of hysteria may be well in a few months. That large class of chronic complainers are incurable hysterics or are the organic cases.

Prognosis depends entirely upon the skill of diagnosis in distinguishing the different forms mentioned, and in our ability to command proper moral and physical treatment of the respective forms. It is always most guarded, but it is probable that the patient in this disease more than in any other may recover promptly.

Pathology.—This will vary from nothing (the first division) to (in the second class of cases) inflammation of muscles, ligaments, or tendons, or tearing of fibres of the same structures. Periostitis, chronic congestion or inflammation of the meninges, and the final passive congestion, or degeneration of the neurones in the spinal cord, or hæmorrhage within the cord may represent the pathological findings in the most serious cases.

Treatment of traumatic hysteria consists in rest cure for several months in the moderate grade, without organic lesion, or of psycho-therapeusis in the most available forms, including hypnotism in these entirely functional cases. The rest treatment is, of course, absolutely essential where any organic condition either of

nervous system or surrounding tissue exists. It may be necessary in some cases, in addition, to procure fixation of the spine by the aid of plaster-jacket or body brace, or by means of head extension with the jury-mast. This is to be prolonged until the parts have healed or are greatly relieved. Counter-irritation over the spine by means of strong tincture of iodine, continued over several weeks, the use of blister or of actual cautery may be of great value. It must not be forgotten that possibly chipping off of a part of a lamina or a vertebral disk may occur, which must be searched for by means of X-rays in all cases, and if found, surgical procedure should be resorted to. These patients ought never be given morphine, else we establish another neurosis, and if drugs are to be used, they should be administered sparingly, the indication being to quiet the central nervous system, in which the bromides, small doses of chloral, hyoscine, and belladonna may be of great service. In the functional cases also change of scene is important, and in any cases where damage suits are pending they should be got rid of at the earliest moment, the physician's duty being simply to give advice from a purely medical standpoint, or upon the witness stand to state his knowledge of the form and severity of the disease from which the patient is then suffering. Much of the discredit upon the profession will be dissipated when physicians learn to give their own opinions after sufficient study in order to know what are the underlying conditions in this unfortunate class of cases.

SALTATORY SPASM

Saltatory spasm is so-called jumpers disease, a hysterical disorder occurring in epidemics in Maine and Canada. It is known as *miryachit* in Russia, and in Java as *latah*. When the patient attempts to stand there are strong contractions of the leg muscles which cause a jumping or springing motion.

SALAAM CONVULSIONS

This is a hysterical manifestation occurring in certain nationalities, particularly the Malay race. The patient usually is in a morbid condition of mind, and without known cause will develop peculiar convulsive seizures, from which he is violently thrown to the ground in a prone position. This will continue for some minutes, or even hours, the patient finally becoming exhausted and passing into a rigid contraction of the general musculature.

WRITER'S CRAMP

SYNONYMS: *Artisan's palsy; Ironer's cramp; Occupation neurosis*

The above are names given to allied conditions, usually of a localized nature, affecting the muscles of a part in spasmodic contractions, associated with more or less pain along the nerve-trunks, and frequently accompanied by wasting of the muscles involved. There are several types of the disease, depending upon the physique of the person affected and upon the nature of the occupation, as above indicated.

Causes.—The condition is fundamentally a form of nerve exhaustion, and is probably central in the majority of cases, the neurones controlling particular movements being exhausted as in neurasthenia. The main predisposing cause is heredity as to neuroses, and secondarily, confinement and overwork, such as the constant application and overwork of a group of muscles, especially when in a constant cramped position. To this primal pathogenesis must be added the irritation produced by the variety of metabolites formed in excess or improperly eliminated. Leucomaines may be complicating factors. Some cases seem to be almost entirely local in nature. These are the cases in which the central nervous system is not called upon for a proportional expenditure.

The more localized types of occupation neuroses may be divided into *muscular, neuritic* (neuritis or not), and *arterial,* depending upon duration and anatomical structures most injured. There are no absolutely fast dividing lines between these latter forms.

Muscular Cases.—This comes on from sudden rather violent overuse of muscles, as in cases of brakemen or motormen. A subacute myositis is very likely set up in these cases. They are usually of shorter duration than the other forms, and abate with the removal of the cause—i. e., with rest of the affected muscles.

Neuritic Cases.—Here we have a peripheral neurosis, which may pass on to a subacute peripheral neuritis. This class is manifestly more severe than the other forms. Symptoms consist of tenderness along nerve-trunks, accompanied by spasm of the muscles when attempting to use them, especially in the occupation which developed the neurosis. Pressure along the nerve-trunks, as is seen in telegraphers, who rest their elbows on the desk, will aggravate the condition. A continuing numbness of the affected part is another prominent symptom.

Vascular Cases.—That these should be given as a type *per se* may be considered dubious from a neurological standpoint, but undoubtedly the arterio-capillary involvement is the primogenesis of a number of cases, and the classification seems at least to be warranted clinically. In these cases the *nervi vasori* and vaso-motor nerves are irritated. In such cases there are none of the cardinal symptoms of the ordinary cases of writer's cramp, no severe spasm or paresis of muscles, but paræsthesia of fingers and forearms. Finally, along with delayed sensations of all forms (but little tenderness), there is particularly a clamminess of the parts so affected. These cases are distinguished from acroparæsthesia by the fact that there is evident pressure cause for the peripheral irritation and from the fact that marked arterial sclerosis is found wanting. The arterial cases may also be divided into the neurasthenic and non-neurasthenic.

The muscular class of cases are the more frequent, and from the fact that the muscles are more affected; and in which treatment is the more prompt in producing results. The prognosis, therefore, is much better in this class of artisan's palsy.

The neuritic class are the usual cases described in classical articles on the subject, and are the most persistent ones, in which there is almost constantly a background of neurasthenia complicating. Such patients are usually emaciated and suffer from insomnia and anorexia. Morris Lewis has best described this disease.[1]

Prognosis.—In the vascular cases this is good in the absence of neurasthenia; and the patient will recover, as a rule, with the general upbuilding of health after the occupation has been abandoned. In a study of some 50 cases of artisan's palsy, at the Infirmary for Nervous Diseases, occurring over some twenty-five years, the writer made a study of the Remote Results of Artisan's Palsy [2] with the following conclusions: That with the proper kind of treatment, and if persisted in, these cases offer a better recovery than was formerly supposed. Two to five years seems to be a fair time for cure in the average case of neuritic type. Where the central nervous system is involved we have the terminal stage of irritation (neurasthenia), most unpromising for complete cure. Muscular types offer the most favourable prospect for ameliora-

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[1] The Neurologic Disorders of Writers and Artisans. Pepper's System of Medicine, vol. v, pp. 504–542.

[2] University of Penna. Medical Magazine, May, 1897.

tion; here no remote conditions need be entertained, and stopping the progress of the disease will generally produce rapid recovery. The hybrid cases I have designated arterial type are also very persisting. In proportion as arterial sclerosis is absent and external irritation is prominent will the hope for complete recovery be gratified within six months to a year under the proper kind of treatment. Where the disease lasts beyond a year, the remote effects are arterial sclerosis, probably induced by the vaso-motor irritation of such long duration; then the deposit of lime salts will of course continue with some remissions, as is the history of this form of arterial degeneration.

Treatment.—The use of antirheumatics, such as potassium iodide, the lithic salts, and sodium salicylate, are of great advantage in those cases where rheumatic diatheses prevail. The use of nitroglycerin or ergot will be of value in the arterial cases with constriction or relaxation respectively through action upon the vaso-motor nerves. Nutrient drugs such as cod-liver oil and the hypophosphites will be of help in cases associated with mal-nutrition. Fresh air and sunlight should be insisted upon in all cases, also relaxation from overwork. We should employ hæmatics, such as Blaud's pills, in cases of anæmia. Since the psychology of the disease is to produce a peculiar morbid depression, it is important for the physician to gain the confidence of the patient and stimulate him to the thorough realization of the need of careful treatment. The local measures of value, which should be continued for many months, are massage, galvanism, and hot local bathing to the part from three to five minutes daily, in cases where there is considerable pain or spasm. In some cases a splint should be applied to the affected member in order to afford relief of spasm by enforced rest. Treatment of existent neurasthenia is absolutely essential for success.

CHAPTER XVII

VASO-MOTOR AND TROPHIC DISORDERS

SYNONYMS: *Parry's disease* (1825); *Graves's disease* (1835); *Basedow's disease* (1840), *etc.*

EXOPHTHALMIC GOÎTRE

THIS is a disease characterized by rapid heart, exophthalmos, enlargement of the thyreoid gland, and by disturbance of the vascular system through functional disease of the sympathetic nervous system.

FIG. 66.—EXOPHTHALMIC GOÎTRE. PROMINENT EYES AND THYREOID GLAND. (Howard Hospital.)

Causes.—There are various theories for this affection. That it is a functional disease of the sympathetic nervous system is held by most authorities. Exciting causes are overwork, fright, auto-intoxication, or infection. It is closely allied to simple goître, or at least hybrid cases frequently are found. Whether or not disturbance of the thyreoid function has primarily to do with Graves's disease is still debatable.

Those contending for this consider that there is an excess of secretion (hyperthyrea), as against the lack of that secretion as a cause of the development of myxœdema (athyrea). The age of onset is usually between the twentieth and thirtieth years, and it is sometimes seen in several of a family. Some observers consider that the affection is due to a lesion in the oblongata.

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Symptoms.—There are two types of this disease, namely, the *acute* and the *chronic.* The former type may develop with great rapidity, following an attack of vomiting and diarrhœa, tachy-cardia, and throbbing of the arteries. Such cases may die in the attack as early as the third day of the affection. Marked cerebral symptoms may be present in this form. More frequently the grad-ual onset prevails, the three cardinal symptoms varying in order of onset. The cardiac and vascular symptoms are usually first to develop, the patient complaining of palpitation and dyspnœa. The pulse is found to be increased in force, the apex beat is in the nor-mal position, and the carotids and abdominal vessels may be throb-bing visibly. Occasionally capillary and venous pulsation may be seen in the hands. At its height the pulse rate may be from 140 to 160, or even more in some cases. It seldom drops below 95 per minute. Emotion tends to increase the heart action, and on exposure of the skin on the chest a transient hyperæmia quite fre-quently can be seen. Basic murmurs are not unusual. Long-standing cases may present cardiac hypertrophy. The heart sounds become greatly accentuated, some cases having been record-ed where the heart-beat could be heard several feet away from the patient. Usually exophthalmos follows next, and is readily recog-nised by protrusion of the eyeballs, and also by the lids not com-pletely covering the sclerotic coat. The protrusion may sometimes cause semi-dislocation of the eyeballs. Vision usually remains in a normal state. Von Graefe's sign, consisting of the inability of the particular lid to follow the downward movement of the eye-ball, though striking, is not a frequent symptom. Stellwag's sign consists in a greater width of the palpebral aperture than in health owing to the retraction of the lid, this usually being found. The patient winks infrequently. Occasionally there is lack of convergence of both eyes. The optic nerves are seldom affected, but pulsation of the retinal arteries is common. The thyreoid swelling usually appears with the exophthalmos. It is generally symmetrical, but seldom as large as in ordinary goître. The blood-vessels are much dilated, and the gland is occasionally seen to pulsate; in either instance a thrill may be felt, or on auscultation we discover a bruit or even a loud systolic murmur. *Motor* symp-toms consist of fine general tremors. The patient becomes anæmic and emaciated. He may be slightly feverish at times. Vomiting or diarrhœa may exist and are usually paroxysmal in occurrence.

Great complaint generally is made of throbbing sensations in the head, accompanied by a sense of flushing and heat with profuse perspiration. The skin will be found, when carefully examined, to be pigmented, favouring Addison's disease; or patches of leuco-derma, or atrophy of pigment and urticaria may exist, as may also areas of solid œdema. Myxœdema has been found to coexist with this affection. The patient is also of irritable temperament and of changeful disposition, mental depression very frequently occurring. Acute mania may develop. A sense of giving way of the legs, a muscular weakness, may exist. There is great diminu-tion in the electrical resistance, which may be due to moisture of the skin from vaso-motor dilatation. Bryson has noticed that the chest expansion is greatly diminished. Breathing is hurried and shallow. Albuminuria and glycosuria are not usual symptoms found present upon frequently examining the urine. Polyuria, hyperidrosis, and œdema are usual. Subnormal temperature might occur.

Prognosis.—This disease usually lasts for many years, but after six months' time it may disappear. Complete recovery is, as a rule, very rare. Instances are recorded of the acute form recovering within the same period of time as its development—a few days.

Pathology.—This is inconstant. The heart is often dilated. Fatty change of its walls may be present. The lobes of the thyreoid are large, firm, and pulpy. Colloid degeneration and cyst formation may be found. The vessels are thickened and athe-romatous. There is proliferation of connective tissue through-out the gland. In the bulb and cord hæmorrhages have been found.

Treatment.—This consists in rest, the absence of worry, abundance of sunlight and oxygen; the use of galvanism, placing the positive pole over the apex of the heart and a Y-shaped divi-sion of the negative pole upon each side of the neck over the cervi-cal sympathetic, with from 3 to 10 milliampères, once or twice weekly, has proved of value in our hands to lessen the rapidity of the heart. The use of strophanthus in continuous dosage is of advantage for the cardiac irritability; where there is great vaso-motor disturbance prominent, the use of digitalis or its products, especially digitaline ($\frac{1}{8}$ grain t. i. d.), will prove of value. The alterative and hæmatinic effect of arsenic, especially when com-bined with iron, will prove a boon in anæmic cases. Ergot is

recommended by some writers. Tincture of belladonna will very frequently give relief, and should be pushed to the physiological limit. Resort to the rest-cure may be a necessity in the worst of cases. The application of Leiter's tube, or an ice-bag over the heart or lower part of the neck, frequently gives relief. Organo-therapy has not been successful in the majority of cases. Surgical treatment cannot be recommended, although several recoveries have already been reported after excision of the cervical sympa-thetic or part of the gland itself.

GLASS-BLOWER'S DISEASE

This is a vaso-motor neurosis, due primarily to sudden increase of blood pressure and the forcing into the blood an excess of oxygen caused by the patient blowing into the blow-pipe, fol-lowed by a deep inspiration when the mouth is removed, at which time the blood becomes overaerated; the latter being the real cause of the cardinal symptoms of the disease. The former is productive of emphysema of the lungs, which, therefore, is a common com-plication. Dizziness is a marked symptom, and is an exaggeration of what can be produced by respirations deeply and rapidly taken during health. In these patients there is a beginning hypertrophy of the right ventricle, accentuation of the second aortic sound, and an increased pulse volume and rate. There soon results ex-haustion, dyspnœa, and a rapid running pulse, the patient com-plaining of fulness of the head, and while exhilarated is unable to co-ordinate properly. This is due to overstimulation of the central nervous system. Mental anxiety is pronounced.

Pathology.—This is, as indicated, overoxygenation of the blood primarily, with sequent hypertrophy of the heart and vascular dis-ease in chronic cases.

Prognosis.—When once firmly established the prognosis is ab-solutely bad if the patient continues the occupation. These cases very seldom live after fifty-five years if they continue work at the same trade. If the occupation is stopped, it may be that the patient may live quietly in comparative comfort for some years.

Treatment.—The patient should quit glass-blowing. Employ antispasmodics, such as lobelia, where the emphysematous symp-toms are predominant; nitroglycerin to steady the circulation and overcome dilatation of the peripheral vessels. It must be re-

membered that there is a possibility of cerebral hæmorrhage in cases with arterial sclerosis. Here the treatment is directed to apoplexy. These complicated cases are generally fatal.

SUDDEN CHANGES IN HAIR

This rare trophic condition occurs, as a rule, as the result of grave mental crises in life or extreme fright.

Symptoms.—The patient after severe psychic shock, as indicated, may notice within a few hours to several days a sudden graying of the hair in streaks or in entirety in a given area; usually of the scalp and eyebrows. In some cases an alopecia will precede, accompanied by erythema or with distinct dermatitis in the area

Fig. 67. — Trophoneurosis of the Hair, showing Change from Dark to White. (Author's reported case.) [1]

affected; and when the hair returns the absence of colour will be noted. The hair, as a rule, remains without colour (gray or white), and may be disposed to fall out from time to time, depending upon the mental condition of the patient as well as the nutrition of the body. The hair change may result in universal and permanent alopecia alone (Fig. 68).

Causes.—The writer has records of two cases produced by shock, one in a male, communicated personally by Dr. E. Pearce, of Ohio. Here the hair turned permanently gray in one night, the man having been frightened during the civil war by rapidly fired

[1] American Medicine, Dec. 28, 1901.

cannon. Another case un-
der our care is of a young
woman, following a dis-
appointment in love, the
hair having dropped out
and then shortly regrew,
being perfectly white (Fig.
67). Psychic trauma is
the cause.

Pathology.—This is in
an indefinite state, consist-
ing in the absence of de-
posits of colour matter,
which must be dependent
upon some trophic disturb-
ance of the nervous sys-
tem similar to other dys-
trophies. Metchnikoff has
described the amœboid ac-
tion of certain leucocytes
which he designates pig-
mentophytes.

Prognosis. — This is
good so far as the health
of the patient is con-
cerned. The hair seldom
returns to its normal col-
our again.

Treatment.—Rest and
tonics are essential. Drugs
to stimulate the sweat-
glands and to favour the
deposits of normal colour-
ing matter would be indi-
cated, such as pilocarpine
and iron. The static
breeze as a stimulant to
the surface of the skin is
indicated. The use of
phosphorus and strych-

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Fig. 68.—Universal Alopecia of Nervous
Origin. (Philadelphia Hospital.)

nine; or the nuclein bodies, or organo-therapy, as thyroid extract, may prove of service in the treatment of this uncommon affection.

HYPEROSTOSIS CRANII (CEPHALOMEGALY)

This disease is allied to osteitis deformans, originally described by Sir James Paget, of London, in a report of the first case, November 24, 1876. When the disease is limited to the cranial

FIG. 69.—HYPEROSTOSIS CRANII. (Author's reported case.) [1]

bones largely, as in the case shown in the two photographs, it is, as already designated, hyperostosis cranii; and becomes a neurological affection only in its relation by contiguity of the enlarged bones to the brain mass.

This affection is a trophic disturbance, involving the bones of the skull, the rest of the bones of the skeleton being but little

[1] Transactions of the Pathological Society of Philadelphia, vol. xviii, 1897.

or not at all involved. There are two types[1] of cranial enlargements:

(a) Upper-head type.

(b) Lower-head type, where the malar bones become involved and produce the leonic appearance. The face and head present an oval with the base up. The bony overgrowth may begin as early

FIG. 70.—SKULL-CAP FROM SAME CASE OF HYPEROSTOSIS CRANII. (See Fig. 69.)

as the twelfth year, and usually it is not later in onset than the twentieth year. The condition is undoubtedly a bony dystrophy.

Symptoms.—The symptoms of this disease are very indefinite. There is usually mental apathy, a state of mental hebetude, and occasionally a condition of what we might call "queer." The

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[1] J. J. Putnam, Trans. Am. Neurological Society, June, 1895.

patient may also suffer from a dull headache. The bony enlargement is gradual, the hearing becomes affected in some cases, and the hairs are noticed to become coarse, and even may fall out to some extent. The case may die of asthenia after many years, or he may be carried off by some intercurrent malady, such as pneumonia, or chronic interstitial nephritis.

Pathology.—This consists of a slow ostitis. The chronic inflammation shows under the microscope irregularly enlarged Haversian systems, atrophy of bone cells, loss of osteophytes and osteoclasts, and the conversion of parts of bone into mere fibrous tissue. On the surface of the bone there is, as a rule, a condensing ostitis. There is frequently cerebral arterial sclerosis in marked degree.

Diagnosis.—This lies between the disease in question and spurious forms of hydrocephalus; but the lack of exophthalmos after prolonged cephalic enlargement with other signs of bone thickness would militate against the latter. The case reported by the writer (Figs. 69 and 70) occurred in a woman seventy-two years of age. Here the skull-cap proved to be $1\frac{1}{4}$ inch in thickness, and as far as I can determine is probably the thickest on record.

Treatment.—The treatment of this condition is, of course, *in cognito.* Nutritional measures and probable benefit from organo-therapy should be thought of. Antisyphilitic drugs should be tried in all cases.

ADIPOSIS DOLOROSA

This is described by Dercum as a disease of adults in which there is a deposition of fat in various parts of the body seen in the form of bunches or nodules, becoming uncircumscribed afterward, and accompanied by pain, diminished sensibility, and great muscular weakness. There is the sensation as of a bunch of worms transmitted to the hand on palpation. Some 15 or more cases have been reported by Dercum, Henry Ewald, Peterson, Loveland, Spliler, Eshner *et al.*

Diagnosis.—This condition must not be confused with myxœdema, since in the former changes do not occur in the hands and feet, and there is an absence of mental symptoms. Ordinary adiposis also affects the hands and feet. It is not painful.

Pathology.—This consists in deposition of fat as described,

in all probability due to a central trophic disturbance. Degeneration of the ultimate nerve filaments has been found by Burr and McCarthy, of Philadelphia.

Prognosis.—This condition may last for many years, the patient dying of asthenia or fatty degeneration of the heart.

Fig. 71.—Adiposis Dolorosa in a Colored Woman. (Medico-Chirurgical Hospital.)

Treatment.—Hygienic measures are to be employed; living in the open air, the use of thyreoid gland extract, and massage may all prove of value by aiding catabolism.

FACIAL HEMIHYPERTROPHY

This is a condition of overgrowth of the soft tissues and bones of one side of the face, being the exact opposite to that of facial hemiatrophy. Care must be observed not to confuse this affection with facial hemiatrophy, in which the normal side would be mistaken for the atrophied side of facial hemiatrophy (see below).

Pathology.—It is supposed to be a trophic disturbance of the central nervous system. The tissues are hypertrophied.

FACIAL HEMIATROPHY

Is a disease characterized by progressive wasting of the tissues, particularly of the bones on one side of the face; starting in childhood, but in some instances at middle life. It usually begins diffusely, but in some cases at a single point on the skin and gradually spreads, involving at first the subcutaneous tissues, then the muscles and bones, and particularly the upper jaw. The atrophy is sharply limited at the middle line, the face looking as though it was made up of two halves from different persons. The colour of the skin changes and the teeth fall out, due to the wasting of the alveolar processes. Even the orbit is seen to become atrophied on the affected side, and the eyeball may become shrunken. There are instances on record in which the disease was bilateral, and in a few cases where the atrophy extended to the back and arm of the same side, the face was also affected. It is a rare affection, only 97 cases being gathered from literature by Sachs, of New York.

Pathology.—In one of the few autopsies made by Mendel he determined the terminal stage of interstitial neuritis in all the branches of the trifacial nerve from their origin to the periphery, more severe in the superior maxillary division. In a case reported by Homen a tumour was found pressing on the Gasserian ganglion.

Diagnosis.—This is not difficult, since the appearance is most striking. Facial asymmetry associated with wry-neck, which, developed in the early childhood, is the only disease with which it may be confounded. The torticollis would make plain.

Treatment.—There is no treatment known at the present time that has a curative tendency in this affection. The hygienic care of the patient should be very strictly pursued as well as supportive

measures, and no doubt the progress of the disease can be prevented to some extent in a few instances.

DERMATITIS GANGRENOSA NERVOSA

Gangrenous dermatitis of nervous origin is a trophic affection, coming on suddenly and periodically. In individuals of neurotic stock it is most commonly seen, and sometimes it is present

Fig. 72.—Dermatitis Gangrenosa Nervosa, showing Scars on Dorsum of
Left Hand of Young Woman, the Result of this Disease.

in patients that are of good health otherwise. The attacks of superficial gangrene in this rare disease the record of the following case will well demonstrate:

A. B., aged twenty-six, white, hysterical, but of good

physique, no organic disease existing at any time. During one night she may develop patches of irregular serpiginous dry gangrene of the superficial layer of the derma upon the hands and trunk. This change is most striking, and may occur during a single night. The sphacelus sloughs off, leaving chronic ulceration, which heals slowly under antiseptic treatment within a few weeks. There is no constitutional disturbance at any time.

Pathogenesis.—These cases are very difficult to explain, but they must depend upon some transient trophic functional disturbance of the central neuron, with probably an auto-intoxication; the fundamental cause being instability of the central nervous system.

Prognosis.—The patient may live for many years suffering from recurrent attacks, and finally recover altogether.

Treatment.—This is largely prophylactic in that of upbuilding the nervous system (perhaps by rest-cure). Change of scene, outdoor life, and hypernutrition may avail towards preventing attacks.

FIG. 73.—SPORADIC CRETINISM. (Philadelphia Hospital.)

MYXŒDEMA

This is a disease characterized by myxœdematous degeneration of the subcutaneous tissues in youth or adult life. It occurs more frequently in males. The affection is due to disease of the thyroid gland in which there is defect of internal secretion of this organ. When the disease begins in early life the name *sporadic cretinism* is applied; and when it occurs as the result of extirpation of the thyroid gland it is designated *cachexia strumipriva*. Myxœdema occurs more frequently from about the thirtieth up to the fortieth year of

age. It may follow erysipelas, rheumatism, persistent hæmorrhages, or syphilis.

Symptoms are of mental obtundity with the physical sign of enlargement of the body through a universal deposit of myxœdematous degenerative material in the subcutaneous tissues. The patient seems fleshy, but the skin is harsh, dry, and sallow-looking rather than normal in colour. There is no true œdema. The expression becomes blank, the patient complains of great feebleness, the lips and lobules of the ear and eyelids thicken. The intestinal tract is torpid, the secretion of the kidneys is but little. This condition exists for some years without much change from month to month. The face is round, in contradistinction to the face in Paget's disease or in acromegaly. The hands are clubbed and puffy, not " spade-like," as in acromegaly.

Prognosis is good as to improvement, wonderful results being obtained by means of the use of the thyreoid gland internally, this making up for the deficiency in the diseased body.

Treatment.—The extract of the thyreoid gland may be given in grain doses, three times a day, gradually increased until palpitation of the heart may be produced. This must be kept up from time to time as the appearance and weight of the case will indicate.

SCLERODERMA

Is a condition of diffused or localized induration of the skin. There are two types: the *circumscribed*, which corresponds to the keloids of Addison or to morphœa; and the *diffuse*, in which large areas are affected. This disease affects females more frequently than males, and occurs in early middle life, although it is seen in spurious form in babyhood—the so-called *sclerema neonatorum*—by some said to be a different affection. This latter disease has occurred only in the French and German races so far as reported. It may at times be of specific origin.

(1) The form mostly seen in this country is the circumscribed type. The patches range from a few centimetres in diameter to the size of the hand or larger, are waxy in appearance, and to the touch hard, brawny, and inelastic. There may be a precedent hyperæmia. Following these manifestations pigmentary changes of the skin occur either of excess or absence—*leucoderma*. The sensory disturbances are rare. Perspiration is diminished or absent. This

type is much more common in women than in men, and is situated about the neck and sometimes along the courses of the nerves. Patches may develop with great rapidity, and may last but a few days or persist for years.

(2) The diffuse form, though less frequent, is more serious. It appears in the face or in the extremities. The patient notices that the skin is unusually hard or firm, and there is a sense of tenseness in making accustomed movements. Gradually a brawny induration develops, the skin becomes united to the subcutaneous tissues, and it cannot be picked up or pinched. The skin may look natural, but more commonly is glossy, dry, and unusually smooth. This form is apt to affect, in order, the upper extremities, the trunk, the head or face, the upper portions of the lower extremities, and least frequently may become universal. Occasionally sensory disturbances are found, the disease may last many years and recovery may occur, or rarely it may be arrested. Rheumatism and endocarditis may complicate, or it may be associated with Reynaud's disease as reported by Mackenzie. The patient may be carried off by pulmonary or renal disease.

Pathology of the disease is unknown, probably a tropho-neurosis dependent upon changes in the arterioles of the skin, leading to connective-tissue overgrowth.

Sclerodactylie is the same disease manifested by symmetrical involvement of the fingers, which become shortened, deformed, and atrophied. Ulcerations have been met with, also great deformity of the nails. This disease is occasionally associated with scleroderma proper.

Treatment consists in mixed luetic medication and in thyroid therapy.

AINHUM

This is a rare condition occurring in the negroes of Brazil, India, Africa, and occasionally in the Southern States. It is confined to the toes, usually the little toe. It commences as a furrow on the line of the digito-plantar fold, which gradually deepens, the under side of the toe enlarges, usually without inflammation or pain, and the toe drops off. The condition may last for years, however, before spontaneous amputation follows. *Treatment* has proven futile in all cases. Antisepsis would be indicated.

ACROMEGALY

This is a distrophy characterized by abnormal enlargement chiefly in the bones of the face and extremities. The word was introduced by Marie,[1] and means large extremities. The disease occurs as frequently in both sexes and usually begins about the twenty-fifth year, although it has occurred much later. Syphilis, rheumatism, and the specific diseases have preceded the development of this malady, but probably have no special connection with it. Disease of the pituitary body is said to be causative.

Symptoms.—In a well-marked case the signs are characteristic. The hands and feet are greatly enlarged; fingers are sausage-shaped. The joints are freely mobile. The hypertrophy is general and gives a spade-like character to the hands. The wrists are frequently enlarged, the arms rarely affected. The nails are broad and large. The lips and tongue are enlarged. The head is increased in volume (oval, with base down); the face more so in proportion, which latter becomes elongated and enlarged in consequence of the increase in size of the superior and inferior maxillary bones. This was especially noticeable in a case coming under the writer's care, which was also accompanied by bitemporal hemianopsia and mental stupor. The lower jaw in particular enlarges and frequently protrudes beyond the upper. The alveolar processes are widened and the teeth separated. The eyelids are at times thickened, the ears greatly enlarged. At times the tongue becomes hypertrophied, and later in the affection kyphosis may develop and the bones of the thorax may slowly enlarge. With it all the skin may seem quite normal, but occasionally may become coarse and flabby, never having the dry, harsh appearance of the skin of myxœdema. The patient is depressed in spirits and physically weak. There may be hyperidrosis, loss of sexual power, frontal headache; or bitemporal hemianopsia due to ventral pressure of the enlarged pituitary body upon the optic chiasm.

Pathology.—In addition to hypertrophy of the bones great cystic enlargement of the hypophysis (pituitary body) has been found. The disease is no doubt a nutritional disturbance analogous to myxœdema, and is probably due to disturbance of function of the pituitary body. Organic change in nerve tissue is absent.

Diagnosis.—The congenital progressive hypertrophy of trunk,

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[1] Revue de Méd., 1886.

FIG. 74.—ACROMEGALY IN A WOMAN, SHOWING ENLARGED SUPRA-ORBITAL RIDGES, INFERIOR MAXILLA AND HANDS. (Medico-Chirurgical Hospital.)

arms, or legs—the so-called giant growth—is easily recognised. In this there are never eye symptoms.

In the osteitis deformans of Paget the shafts of the long bones are involved; and in the head the bones of the cranium, but not of the face, as Marie states. In Paget's disease the face is triangular, with the base up; in acromegaly it is ovoid or egg-shaped, with the large pole down; while in myxœdema it is round. In hypertrophic pulmonary osteo-arthropathy, while enlargement of the hands and feet occur it is chiefly in the lower three-fourths of the forearm and legs, but there is not any involvement of the face. (See below.)

Prognosis is bad as to cure. Sometimes the patient can be bettered by general hygienic *régime*. Thyreoid extract and pituitary body have been recommended, and much good has resulted from their use. Alteratives, hot baths, rest, and sunshine may tend to stay the process.

The liability to sudden death must be remembered, as in the instance of a typical case under the writer's observation. The man apparently died of heart failure of reflex origin, he having fallen over while at work, and when in average physical health. No autopsy was permitted.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Marie has given the name hypertrophic pulmonary osteo-arthropathy to a queer disorder characterized by enlargement of the hands, feet, and of the ends of the long bones, chiefly of the lower three-quarters of the forearms and legs. The bones of the skull and face are not involved. The terminal phalanges are much spread with both transverse and longitudinal curves. The nails are also enlarged and much curved over the ends of the phalanges. Scoliosis and kyphosis have both been met with. It is a chronic disease, and in nearly all instances has been associated with some long-standing affection of the bronchi, lungs, or pleura—hence its name—of which sarcoma, chronic bronchitis, and pulmonary tuberculosis have been the most frequent. It may develop in those who have had syphilis. It is more common in adult males. Thornburn [1] has collected some 30 cases of this rare disease and

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[1] British Medical Journal, vol. i, 1893.

others have been reported since. We have seen 3 cases. Dull pain in the extremities was a symptom in each patient.

Pathology.—This is very obscure. Marie suggests that the toxins of the pulmonary disease are absorbed and irritate bony and articular structures. Thornburn believes it is a benign chronic tuberculous affection. The nervous system may be at the bottom of the disease; it is very probably a trophoneurosis.

Treatment should consist in combating the pulmonary disease. The use of cod-liver oil, hypophosphites, and nutrients may thus be of service. Protection of the parts from cold is essential. Ichthyol ointment (1 dram to ½ ounce) may have some soothing influence through its sorbefacient effect. Antisyphilitic treatment should be carried out faithfully.

ARTHRITIS DEFORMANS (Rheumatoid Arthritis)

Ætiology.—Dr. John K. Mitchell, Sr., contended that this was a trophic nervous disease as long ago as 1827. There seems no better place to classify this disease. It is a trophic affection involving the smaller joints of the hands and feet; occasionally the larger joints. It is characterized by changes in the cartilages and synovial membranes, with periarticular formation of bone and great deformity (see Figs. 75, 76, and 77). The association of the disease with shock, worry, or grief will support the theory of its nervous origin; also its similarity to arthropathies, as of tabes. The symmetrical distribution of the lesions, the skin and nail changes, and muscular wasting out of proportion to the joint mischief point towards a trophoneurosis.

Ord regards the disease as similar to progressive muscular atrophy due to *primary* changes in the cord; or to peripheral irritation, as of trauma or uterine disease, these causing *secondary* changes in the cord. Females are much more liable to the disease than males. In Garrod's table of 500 cases it occurs in females 411, and in males 89 times. Exhausting disease, such as prolonged sepsis or typhoid, may precipitate it.

Symptomatology.—The disease may begin in children (six to twelve years), in old age but rarely. It usually originates between twenty and thirty years of age. Hereditary influence favours early development. It has no more predilection for the poor than the rich, and I have seen it in the most lowly as well as in the

best stock in America; though in England and on the Continent
the poor, from given statistics, suffer most. There are three
forms of the disease: (1) The general progressive form; (2) the
partial monarticular form; and (3) that in which Heberdeen's
nodosities are prominent. In the latter form " little hard knobs "

Fig. 75. — Agglutination, Patella to Tibia and Subluxation of Knee, in
Rheumatoid Arthritis. X-ray photograph (kindness of Dr. R. P. Cummins,
Howard Hospital).

develop gradually at the sides of the distal phalanges. These occur
later in life in the average case (thirty to forty years or over).
Such subjects may have had digestive troubles. The joints may at
first be swollen or red, especially when injured. The dorsal sur-
face of the second phalanx increases in size, giving the character-
istic appearance of the joint. Charcot contended that urate of
soda is never - deposited. These patients seldom have involve-

FIG. 76.—EROSION JOINTS OF HANDS. RHEUMATOID ARTHRITIS. X-ray photograph (kindness of Dr. J. C. Boggs, of Pittsburg).

ment of larger joints, although exceptions do occur. (See Figs. 75 and 77.)

Of the *progressive* form there are two types, *acute* and *chronic*. Many joints may be involved at first in swelling, redness, distention of synovial sheaths and bursæ. It is more common in women between twenty and thirty and after prolonged lactation or rapid childbearing. Remissions may occur. The *chronic* form is the most frequent. Pain on movement and slight swellings due to effusion into joints and the periarticular sheaths are the first symptoms. Pain is, however, an extremely variable symptom. Gradually the shape of the joints is greatly altered, partly by the great thickening of the ligaments, and still more by retraction and atrophy of the muscles.

Crepitation can be felt on movement due to eburnation of the articular surfaces. The joints finally become completely locked, not by true ankylosis, but by the osteophites, which, as Osler describes, are like the ring bones in horses. Spurious ankylosis may occur also from thickening of the capsular ligaments and by fibrous adhesions. Contractures and contractions occur and bring about most marked flexion of legs upon the thighs and the thigh upon the abdomen. In other cases the muscular wasting is so rapid that central disease will again alone explain their pathology. Fortunately in some cases the fingers and toes are not so much involved, and the patient is able to knit, etc. Numbness, tingling, pigmentation, and glossiness of the skin exist. *In the worst cases all the joints of the extremities become locked and the patient lies curled up in bed helpless.* Conservatism of nature and compensatory function are beautifully demonstrated in some of these pronounced cases, however; the patients frequently accomplishing under great difficulties many artistic designs where the talent exists.

The *partial* or monarticular forms are found mostly in the aged, and are particularly seen in the knee, hip, shoulder, or spinal column (spondylitis deformans). (See Rhizomyelique Spondylitis.) If in the hip, the term *morbus coxæ senilis* is applied to the affection. Injury frequently precipitates cases involving one joint. This form occurs particularly in men. We have one such case.

Diagnosis.—Arthritis deformans should not be confused with rheumatism or gout. The monarticular form of simple local

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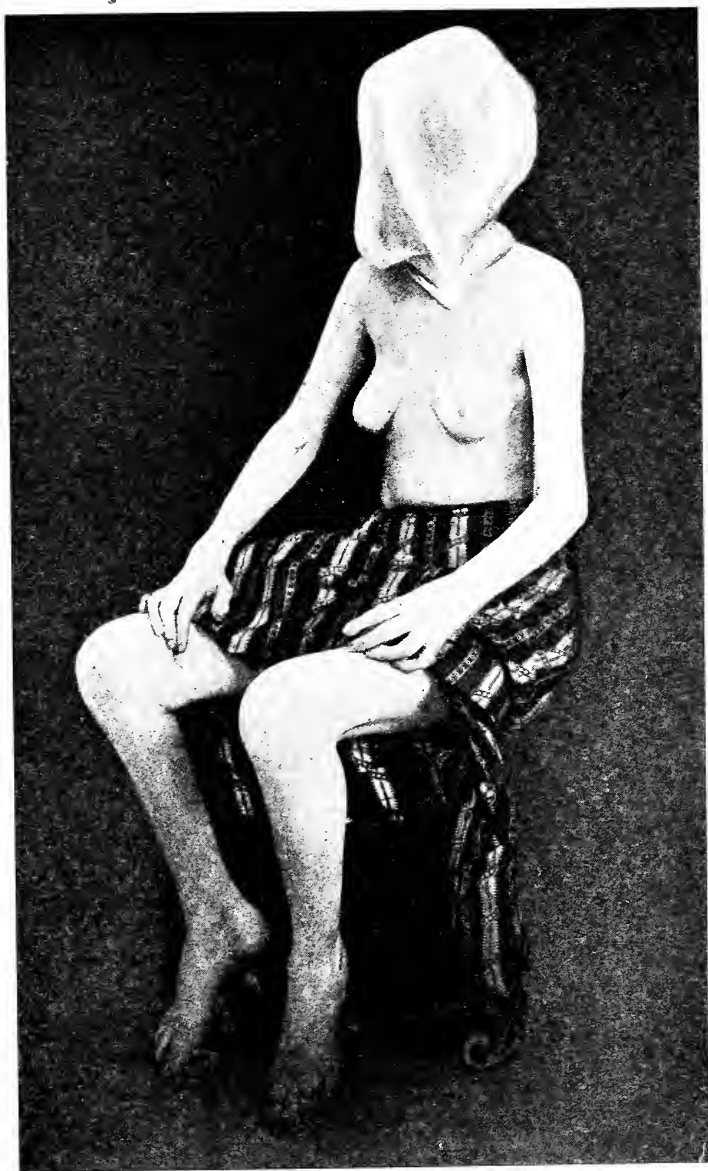
Fig. 77.—Rheumatoid Arthritis with Contractures. Bedridden girl before operation. (Howard Hospital.) (See Fig. 78.)

FIG. 78.—Rheumatoid Arthritis, after Operation and Braces are Applied. (Howard Hospital.) (See Fig. 77.)

arthritis is characterized by more pain than in arthritis defor-
mans, also by thickening of the capsule and the ligaments, and if
neuritis exists with it, a greater wasting of the shoulder-girdle
muscles.   Arthritis deformans also differs. from chronic rheuma-
tism in the existence of extensive structural alterations especially in
the cartilages, according to Adams.   *Pathologically*, too, the
changes in the joints differ essentially from those of gout in the
absence of deposits of urate of soda.   No definite microscopic
change has been found in the spinal cord in arthritis deformans,
although there must be some morbid physiological process here as
a causative factor of this singular affection.

Treatment.—It is an incurable disease.   Much can be done
towards alleviating the sufferings, however, or to arrest its prog-
ress.   Iodide of potassium or sodium, quinine, the salicylates, and
arsenic are all valuable.   The use of tonics, and particularly the
hypophosphites, is of value in supporting the system and in sup-
plying the waste of salts from the nervous tissue.   Syrup of the
iodide of iron I have found one of the most useful drugs to cor-
rect anæmia, and with restored blood pabulum the progress of the
disease is often stayed.   Morphine in small doses by the mouth
often has a favourable effect upon pain. and in some unknown ·
fashion may tend to check the affection.   Bloodless operation of
extension, or a tenotomy, are means to help these people to get
about on their feet.   The accompanying illustrations show well a
girl who had been bedridden for two years, a helpless cripple.
She now is able to walk about alone, first having had hyperex-
tension and tenotomy performed by Dr. C. H. Frazier, then casts
applied, and finally braces adjusted.   The ankles and feet were free
of disease.   The back cases offer little help from the surgeon.

HEAT EXHAUSTION

This is a collapse induced by excess of heat to the body, induced
through natural telluric conditions, or by artificial heat. as an
overheated room.   It is generally associated, however, with the
state produced by the excess of sun-rays.   The condition heat ex-
haustion is allied to sunstroke, the clinical difference being in the
ætiology—i. e., the heat has not been so intense as a rule or the
patient is more resisting ; and secondarily in the symptomatology
in particular : heat exhaustion being a complex where there is

subnormal temperature of the body, feeble pulse, a status much allied to shock, the patient being attacked rather suddenly.   He complains of headache and faintness, and occasionally he may lose consciousness for a short time.   The duration may be from an hour or so to many hours.   The *pathology* merely consists of a disturbance of the heat centres and vaso-dilatation through the development of toxins, on account of superheating of the body.

Prognosis.—This is good if the patient is not re-exposed to the heat, and fatty heart or nephritis are absent.

Treatment.—This is the same as in shock—ammonia, strychnine, and quietude.

SUNSTROKE

Thermic fever; Heat-stroke; Coup de soleil; Insolation

This is due to a more intense or more prolonged application of heat to the body, as seen under the heading of heat exhaustion. The *symptoms* consist of a feeling of congestion of the brain, muddled thoughts, as the patient will tell you afterward, and a sense of extreme weakness, he usually falling over at his occupation into an unconscious state in this period.   He may pass at once into severe tonic and clonic convulsions.   The temperature rises to 102° or 103° F., gradually increasing until it may reach 109° or more; the highest case authenticated and followed by recovery being 112° F.   Uræmic symptoms usually complicate the disease, due to the fact that the renal circulatory apparatus is disturbed, and occasionally marked transient albuminuria will be found. The duration of the affection is apt to be short and fulminating, the patient either recovering after a few days or death may supervene before this time.   In those cases that recover there is a great liability for relapse or for a chronic meningitis to be set up.

Prognosis.[1]—Cases with prolonged unconsciousness or suffering from a high temperature usually prove fatal in 75 per cent of the cases.   Cases with high temperature and preservation of mental faculties are more favourable.   When there is a failure of the kidney, determined by carefully examining the urine, the prognosis can be put down as also being bad.   In every case there is a great liability to relapse on slightest exposure, and in a great number of

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[1] The statements in this paragraph are made from a careful study of 30 cases at St. Agnes's Hospital in 1896.

cases there is a great liability to heat exhaustion or sunstroke at subsequent periods. Chronic headache usually follows.

Pathology.—There must be some chemical change in the body causing paralysis of the centres; thus more heat is produced than normally.

In those cases that recover there is a great liability for a chronic meningitis to be set up in time, which may be incurable.

Treatment.—This consists in the application of cold, preferably moist cold, in the form of an ice-bath. This should be associated with friction to the extremities with ice. The head should particularly be kept cold in order to prevent aggravation of the cerebral congestion. Antipyretics are of value in sthenic cases. Convulsions must be controlled by the use of chloral and bromides, given *per orum,* or *per rectum* in the instances where the patient cannot swallow. Rectal feeding should also be employed in those cases where the stomach is irritable, and in all cases the food should be simple, such as peptonized milk, etc. The patient would better change his occupation to prevent recurrence.

MYASTHENIA GRAVIS

By this disease is meant the peculiar condition in which the patient loses power, periodically and suddenly, in the muscles without any definite neurological symptoms. It is closely allied if not identical with *periodic paralysis,* as described by Taylor, of Boston, and studied also by J. K. Mitchell, of Philadelphia.

The *symptom* is palsy; the patient on waking in the morning finds that he is unable to move the muscles of certain parts of the body or of the entire voluntary musculature. This may last for a few to twenty-four hours, or even several days. In a very short time the case is perfectly helpless, without any sensory or psychic disturbances, save an indefinite paræsthesia or a mental anxiety not due to brain disease, but to the fear he naturally has of impending death. The patient slowly recovers from the condition, and may go about his duties as usual within the space of a few days, only to be stricken down again at any time. Undoubtedly the underlying condition is an auto-intoxication, either from waste products from within or from absorption from without. Study of the blood has been made, and nothing has been found excepting a mild leucocytosis during the attack. As there are no

temperature symptoms, it is not a condition of sepsis, at all events. It is more likely a failure of the normal chemical changes in the nervous system, and therefore inability to carry on the function of the neuron, although the expression of this seems to be alone confined to the muscles. It should also be mentioned that there is no reaction of degeneration, though the reflexes are absent.

Prognosis.—This is very dubious, and the disease may last for years, the patient dying in an attack or of some intercurrent affection. Ultimate recovery may take place in those individuals where the normal metabolism becomes restored.

Treatment.—This consists in the greatest hygienic care as to proper nourishing, protection from cold and dampness, prevention of overwork, both mental and physical, and particularly to dietetics. The food should be simple, consisting largely of proteids, as milk, eggs, and those substances containing excess of nuclein—beans, peas, etc.—also those containing iron, such as spinach, although the patient should never eat too heartily. During an attack the patient should be kept in bed. Strychnine is given in doses of $\frac{1}{30}$ grain up to $\frac{1}{12}$ grain hypodermically. While venesection would be a dangerous procedure, the use of hypodermoclysis of salt solution or even transfusion, if this can be properly done, would be of great value. This subject has not been sufficiently worked up by the surgeon. It is to be recommended as an ideal treatment in this disease. The use of thyreoid extract, thymus gland, or even suprarenal extract capsule preparations sometimes seem to meet the conditions and probably be of value in aiding metabolism. Bone-marrow is another organic preparation that may be of value. The bromides were valuable in a number of the cases coming under the observation of Holtzapple, which is probably due to restoration of vaso-motor tone in the motor horns of the cord.

FAMILY PERIODIC PARALYSIS[1]

Cases of this rare disease have been detailed by Putnam and John K. Mitchell and others. The pathology of the affection is unknown ; blood examinations have shown no characteristic

[1] The valuable paper read by Dr. G. E. Holtzapple, of York, Pa., at the meeting of the Medical Society of Penna., Sept. 24, 1903, throws more clinical light on these enigmatic diseases than has yet been given to the profession.

changes, nor has the urine. Some undiscovered toxin, probably of autochthonous origin, is the cause; a tendency to degeneration of this sort passing from one generation to the next. (See Myasthenia gravis, from which it differs by being a family disease.)

Symptomatology.—It consists of periodic attacks (of more or less sudden onset) of wide-spread flaccid motor palsy, associated with loss of knee-jerks and electrical excitability, with little sensory or psychic symptoms of any kind. The attacks begin in the majority of cases between the second and third decades of life. In some cases there are prodromata, such as a feeling of weariness, formications, numbness, headache, backache, sweating, and loss of vaso-motor tone. As a rule the paralysis begins when the physiologic ebb is lowest—i. e., at night—and continues for several hours before the acme of the paralysis has set in. The legs are usually first affected, then the muscles of the arms, trunk, and neck in severe cases. The cranial nerves usually escape palsy. During the attack there is loss of knee-jerk and absolute loss of electrical irritability of the muscles, without degenerative reaction, however. But in the interval between these attacks the reflexes and electrical irritability are normal. In some cases during an attack myocardial weakness is found, the heart may become dilated, and hæmic murmurs (usually a mitral systolic murmur) are found. This also disappears with the normal intervals. The patient is mentally apathetic during attacks, but never further affected as to his mind. The usual duration of an attack of transient paralysis is from five to thirty-six hours. Recovery from the attack takes place gradually. Each succeeding attack may become more profound, leaving the patient in a more marked asthenic condition. It is incurable. Treatment during attack should be *hypodermics* of strychnine and digitalis, bromides, warm baths, and *effleurage.* Hypodermoclysis of normal salt solution may aid recovery. Thyreoid should be tried. The disease should practically be managed as is a case of myasthenia gravis, already studied, and from which it differs again in the more pronounced periodicity. (See p. 343.)

CHAPTER XVIII

GENERAL TOXÆMIC DISEASES OF THE NERVOUS SYSTEM

HYDROPHOBIA—RABIES

HYDROPHOBIA, or rabies, is a convulsive disease induced by the virus of a rabid or "mad" dog. The disease primarily must originate *de novo,* more particularly in the canine. The infection is more frequent in hot weather, when the condition of the blood of the animal favours the growth of the germ. As yet this has not been isolated. The name hydrophobia is a misnomer, in the fact that the patient does not have the fear of water, *per se;* but in the symptomatology of the disease, the spasm of the pharyngeal muscles prevents deglutition and makes the swallowing even of liquids difficult or impossible. The usual method of inoculation in the human being is by means of the bite of a "rabid" or delirious dog. As a rule, the wound is slight, so that frequently little attention is paid to the point of infection. Where the wound has been freely opened or cauterized immediately after its inception, the less is the possibility of development of rabies following the bite of an animal suffering from hydrophobia.

Symptoms.—Development of the symptoms of rabies seldom takes place sooner than six weeks or after three months from the date of infection. The patient is suddenly seized with extreme nervousness and excitability; the muscles remain rigid, especially those of mastication and the constrictors of the pharynx; the head is drawn back in tonic spasm. The patient complains of his inability to use his extremities well on account of "stiffness": the mental anxiety apparently progresses out of proportion to the severity of the disease, and in these cases, no doubt, the apprehension of the terrible malady produces a species of hysterical insanity complicating the rabies. Within a few hours the patient may go from the *tonic* convulsion into violent *clonic*

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spasms; and these may be brought on, too, by slight sources of reflex disturbance, such as loud noises, sudden throwing of light upon the patient's sensitive retina, or as in attempting to catheterize the patient, which is sometimes necessary, due to spasm of the sphincter vesicæ; all of these reflex convulsions being the result of excitation of the hypersensitive special sense and general sensory neurones. The patient's mental condition gradually becomes maniacal, and he tosses about the bed in fear and delusion; there is a prominent feeling of impending death due to contraction of the throat muscles and those of respiration, and even the diaphragm; while the cardiac arrhythmia and cramp produce symptoms allied to angina. Any attempt at swallowing, as before indicated, will cause an aggravation of the throat symptoms, although the natural desire to obtain liquids is really nature's effort at producing dilution of the poison in the system, which is defeated by the inability to perform the act of deglutition. Opisthotonus is not an unusual position for the body to assume in the interim of clonic spasm, the trunk rigidity always remaining, with but little relaxation; or the body may be drawn in emprosthotonus or pleurothotonus, depending upon the muscles of the trunk more particularly affected. This sudden cramping of muscles in the various parts of the body causes violent pain, so that when the mentality is not entirely blunted there will be produced evidences of extreme suffering in the face aside from the general spasm of muscles of expression. The temperature rises in irregular fashion to 103° or 104°, and is proportionate to the degree of infection, especially to involvement of cerebrospinal meninges. If the patient does not die of exhaustion in the convulsions at the end of the third day the *paralytic stage* sets in, the temperature drops, the muscles become flaccid, the patient enters into low muttering delirium in profound unconsciousness, showing extensive degeneration of the cerebral cortex; finally he dies of exhaustion, the heart generally stopping before respiration has ceased. Risus sardonicus usually precedes death.

Diagnosis.—The only disease with which it can be confounded is the one we shall next discuss, tetanus, symptoms of which, when given, will fairly accurately set aright this differentiation, which lies particularly in the mode of infection and in the stage of invasion of the acute symptoms. Hysteria may simulate.

Prognosis.—The prognosis of hydrophobia is almost univer-

sally fatal. Some mild cases have no doubt recovered under treatment, just as rarely occurs in tetanus under the most active therapeutic measures.

Treatment.—Treatment is preventive. The antitoxine method discovered by Pasteur is the only measure certain of saving the patient. It should be resorted to immediately upon learning the true nature of the disease in the animal that has been the source of infection; and in view of the severity of the malady, should be done without delay where there is even suspicion of rabies. The antirabies serum should be injected according to the dosage prescribed by the laboratories where it is prepared; that of Gibier in this country being the most desirable product. Treatment after the development of the disease is entirely symptomatic, the use of cannabis indica, of the Calabar bean, the bromides, and chloral being indicated. Guarding the patient from noises or extremes of light or cold is an essential point in amelioration of the terrible symptoms of this, until recently, usually incurable disease.

TETANUS

Lockjaw

Tetanus, or lockjaw, is a disease characterized by violent convulsive seizures beginning in the muscles of mastication; hence the name lockjaw. Consciousness is preserved.

Ætiology.—The cause of tetanus is a short, rod-shaped bacillus, whose habitat is in the earth. For this reason gardeners and hostlers are particularly susceptible to infection. I have in mind the instance of a splinter which produced tetanus when run under the nail of a farmer while in his stable; and another case of a boy who received a brush burn over the tibia while driving mule-cars in a coal-shaft. Penetrative wounds, as by a nail, are also sources of inoculation. The incubation of lockjaw is much shorter than that of hydrophobia, being an average of *ten* days until invasion of active symptoms.

Symptomatology is indicated in the definition. It begins with rigidity of the jaw (trismus); difficulty in swallowing; "stiffness" in the back of the neck, the patient often complaining at the outset of simply an inability to move the muscles well on account of this rigidity. Soon the muscle rigidity becomes generalized, and there is a tonic contraction of the entire muscula-

ture, interspersed with twitchings of the small muscles of the extremities; with this there is great mental anxiety, but the mind remains perfectly clear. The disease rapidly progresses, clonic convulsions occur in which the patient suffers violent pain and cries out in his torture, although efforts at clear phonation are interfered with by spasm of the pharyngeal and laryngeal muscles. There is forcible dejection of urine and fæces, also abortive efforts at vomiting. With sudden noises, as the slamming of a door, or throwing of light upon the patient, the subject will go into violent convulsive seizures of a clonic nature. The patient will be at times painfully contorted in various parts of the body, the picture finally ending with general convulsions, as above stated. There is a continuous tonic contraction during the active course of the disease. There is general hyperæsthesia, and also enormous excitability of the special senses, such as photophobia, etc. At intervals between the serious motor explosions, and even under the effect of enormous doses of depresso-motors, the patient may get a few moments of disturbed slumber, only to be aroused by a sudden convulsive attack. The progress of the case is gradually downward, the patient finally at the end of three or four days going into collapse, with slight relaxation of the muscles and dropping of the temperature below normal. The *algid stage* now supervenes, also *risus sardonicus*, death soon following from exhaustion. [*Cephalic*, or Rose, tetanus is a chronic form of subacute tetanus in which trismus is the prevailing symptom. The course is longer (weeks), prognosis good, and treatment (same as tetanus) usually curative. A case of Rose tetanus under my care, that of a physician, caused by a kick on the nose by a mule, recovered promptly.]

Treatment is as yet but symptomatic and very unsatisfactory, since the cultivation of the tetanus bacillus cannot be grown outside of the human body; also, as we have not been able to produce the disease in mild enough form in the lower animals to avoid death, the antitoxine has not been obtained. With the rapid advances in bacteriology, it is to be hoped that such antitoxine will be produced in the near future, when we will have a prospect of successful treatment of this terrible malady. The treatment at present to be adopted is immediately to open up a wound where from the above pointed-out sources of infection any such wound exists. Even after the tetanic symptoms have developed, it would be wise, though late, to excise such a wound in the hope of staying

further infection from the nidus. The patient suffering from tetanus should be placed in the quietest possible surroundings; the room should be thoroughly shaded and kept entirely quiet from all sources of reflex irritation through the general or special senses; the slamming of a door I have seen the cause of precipitating a convulsion. Chloral is in my experience one of the best drugs at command for the alleviation of convulsion, and while the heart may be depressed, the excito-motor system is so stimulated in this disease that the use of such depressant far surpasses any ill effect upon other organs, as the heart. This drug can be given in 15-grain doses every three hours, and pushed until some amelioration of the convulsive seizure obtains. The remedy can, in cases where the patient is unable to swallow, be administered *per rectum*, and in this case should be given in about twice the dose as when taken by the mouth. Bromides take second place in the management of the disorder, and can be combined with the chloral in cases where the former fail. By such combination we have ideally what H. C. Wood terms the " crossed " action of drugs— that is, the maximal effect upon one part of the system, in this case the depression of the motor centres in the brain, with a minimal effect upon, as in this case, the cardiac and respiratory functions. Morphine should be used hypodermically with caution where the case is in a status of convulsions, the idea being here to quiet the nervous system quickly in order to prevent death through continuation of convulsions and consequent exhaustion. This drug should not be used in the interim, since it is not desirable to block the kidneys in any way. Venesection in tetanus would seem to be rational therapeutics, for by this measure we could hope to carry off the toxine in part, and by hypodermoclysis possibly to dilute the blood pabulum and flush out the toxine from the nerve-centres in the brain and cord. The principal drawback in the procedure would be the tonicity and spasm of the muscles and blood-vessel walls which prevent both the egress and ingress of salt solution.[1] Calabar bean I have seen cure one case. It should be pushed, in the form of the fluid extract, to the physiological limit in cases where the chloral, bromide, and opium treatment outlined has failed to produce any amelioration of symptoms within the first twenty-four hours.

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[1] Normal salt solution is prepared by adding 46 grains of sodium chloride to the pint of distilled water. The water is kept at 110° F.

It should be mentioned that such patients ought to be especially guarded from hurting themselves, for while they are perfectly conscious and appreciate the terrible suffering, they are, of course, unable to protect themselves from injuries resulting from the violent convulsions. The tongue should be particularly guarded from being bitten, and the saliva should be kept clear from the throat, more by the posture of the body (preferably placing the patient on his side from time to time) than by any effort at mechanical swabbing of the throat. The bed should be very large, soft, and placed in the centre of the room, so that attendants can relieve and guard the patient from all sides. If hyperpyrexia should develop, the application of ice-bags to the head and spinal column would be of value.

TETANY (TETANILLA)

Tetany is a subacute or chronic disorder that is characterized by intermittent or persistent tonic contractions beginning in the extremities and associated with paræsthesia and hyperexcitability of the motor and sensory nerves.

Ætiology.—It is a rare disease in America, but quite common in Europe, especially in Austria. It is more common at the second to fourth years of life, and again at the age of puberty, and is very rare after fifty years of age. It is more frequent in males in early life. There is less difference in the frequency of its appearance in either sex after the twentieth year. Rachitis is a predisposing cause. Exciting causes are exhausting influences, like lactation, sepsis, fatigue, mental shock, fevers, or exposure to cold or wet. Dilatation of the stomach and absorption of toxines from the intestinal tract in consequence, intestinal parasites, and alcoholism may also cause the disease.

Symptoms.—The attacks are usually paroxysmal, lasting from a few minutes to many hours. In the continuous cases the spasm may last for days or weeks. There may be a feeling of general lassitude or of numbness or pain in the extremities for a short time before the onset. In the majority of cases the spasms are confined to the hands alone or to the hands and feet. The fingers are closely pressed together, the thumbs adducted and pressed against the index-finger, the so-called *writing posture*; or are flexed into the palms beneath the fingers, the so-called "accoucheur's

hand." The forearm is flexed and upper arm adducted. In the lower extremities the toes are strongly flexed, the knees and feet extended, the feet also being inverted. In bad cases the muscles of the abdomen, chest, neck, and face are involved. Trismus and drawing out of the angles of the mouth give a peculiar physiognomy to the patient (risus sardonicus). Opisthotonus and dyspnœa may result. The orbicularis oris and extra-ocular muscles may develop contractions, closing the eye, thus adding a squint to the patient's grotesque appearance; the muscles of the larynx (laryngismus stridulus), œsophagus, and bladder may be affected. There is no pain unless the spasms are severe. Fibrillary tremors may be seen in the contracted muscles. There may seldom be loss of consciousness, and this usually occurs in the cases associated with extreme debility or gastrectasia. Usually the mind is quite clear. There may during attacks be hyperæsthesia of the skin of the parts affected. The spasms may arouse the patient from sleep. Fever is at times present in epidemic form. In such there must be some mixed infection complicating the case.

(A) In the intervals between attacks continued pressure over a nerve-trunk may bring on spasms (*Trousseau's sign*). Even a slight tap over a nerve with the plexor may induce spasm of the muscles supplied by it. Thus tapping over the facial nerve at its point of exit from the stylo-mastoid foramen will produce contraction of the muscles of the face, particularly of the lips. This is called the facial phenomenon or *Chvostek's sign.*

(B) Electrical excitability (*electrotonus*) of the motor nerves and muscles is increased, a feeble current causing tetanic contractions. Instead of the normal formula we may have AnClC or AnOC > KClC. This is called *Erb's symptom*. There may be a cathode-opening tetanus, a phenomenon not found in any other affection. Gowers says there may be a reversal of the polar formula. Thus a positive-pole opening contraction occurs earlier than a positive-pole closure contraction.

(C) *Sensory nerves* are also more *irritable* to mechanical and electrical stimuli; a weak current passed through such a nerve as the supra-orbital may cause paræsthesia in the parts to which the nerve is distributed. This is called *Hoffman's symptom*. The auditory nerve reacts easily. In exceptionally mild cases the symptomatology of tetany may be of paræsthesia and stiffness of muscles without real spasms. In this latter form Trousseau's symp-

tom cannot be elicited. Infantile tetany is less apt to be severe than that in youth or adults. Symptomatic tetany from brain disease must be differentiated from true tetany principally by the presence of the brain disease and trismus.

Duration.—When the spasms are continuous the disease lasts but a few weeks, but when they are intermittent they may last for months, as though it took a certain time for the toxine to be eliminated, and in which the spasm assisted in some unknown fashion. (Epilepsy may also be bettered by an occasional attack in toxic cases.)

Tetany usually lasts a few weeks or several months. It is liable to recur on a return of the exciting cause.

Diagnosis.—The disease is usually told by the symmetrical distribution, the hyperexcitability of muscles, motor and sensory nerves. Trousseau's sign is found in no other affection; also it is very rare to find the " facial phenomenon " and the electrical and mechanical irritability of such quality in any other disease. The acoustic-nerve irritability is also characteristic. Tetany rarely causes death. *Tetany is distinguished from tetanus* by the intermittent tendency of the spasms, their feeble character, and the fact that they begin in the extremities and extend to the trunk; also the absence of trismus except very late in the disease. It may recur annually for a number of years.

Pathology.—There is a congestive and irritative condition of the gray matter of the spinal cord. In epidemic tetany there must exist some infectious poison. In other cases mucin circulation in the blood is found, as in that type due to thyreoid disease (*thyreoid tetany*). Rheumatic poison causes other cases of tetany. Ergot may produce symptoms resembling tetany. In infantile tetany the irritation is due to cortical affection, to meningitis, rachitic poison, or to reflex irritation from disorder in the gastro-intestinal tract. The latter scarcely ever happens in adults. Tetany is a symptomatic disease, but the primary causes are so many it deserves the distinction of a name on clinical grounds. There is no *definite* pathology, no characteristic constant lesions being found at autopsy.

Treatment.—Stop the cause, having searched most carefully for it. In cases following the removal of the thyreoid or due to disease of that organ, thyreoid extract should be given. Lactation must be stopped in adults. Diarrhœa, indigestion, or rickets

should be cured, or removal of intestinal parasites if existent. Symptomatically: Rest, nourishing food, tonics; and for the spasms, bromide of sodium in 1-dram to 3-dram doses combined with chloral for its "crossed action" are indicated. Hyoscine hydrobromate, grain $\frac{1}{100}$ t. i. d., may be of service; or valerian or other antispasmodics may be employed. Warm baths are of service. Digitalis is of value in nocturnal tetany, as advised by Gowers. This dose given at bedtime very likely acts through toning up the circulation and hastening elimination of the poison. The anodal pole of a weak galvanic current may allay the irritability for a time. Inhalations of chloroform or ether may be necessary to control the convulsive seizures.

CHAPTER XIX

DRUG INTOXICATIONS

ALCOHOLISM—ALCOHOLIC INSANITY

This is a disease produced by indulgence in spirituous liquors. There are two sorts: *acute* and *chronic*. The acute is usually brought on by the excessive indulgence at periodic intervals, the chronic by the result of more or less constant imbibition. The development of these conditions will depend more or less upon the idiosyncrasy of the particular individual. Those forms of alcohol containing fusel oils are more apt to precipitate an attack.

Symptoms of Acute Form.—The patient complains of confusion of ideas, is mentally excited, the sensorium, however, generally being obtunded, so that exposure to cold, for example, is not well appreciated. There will usually be fine tremor of the extremities, lips, tongue, in the acute alcoholic state. The urine will be found to be of a high specific gravity and often loaded with uric acid, especially with those who have a lithæmic diathesis. The patient will complain of a full feeling in the head, often of palpitation of the heart and symptoms of acute indigestion, such as nausea and vomiting, followed by a thin and watery stool. This syndrome of symptoms is followed by general muscular weakness and exhaustion. The clinical picture may last from a few days to many weeks, depending upon the continued indulgence in alcohol. At any stage of the disease the patient may develop *mania a potu* or delirium tremens, which is characterized by great mental and physical excitement, by hallucinations and delusions, particularly of seeing weird objects (the seeing of snakes), this being rather a constant illustration. The patient also hears uncanny voices or may describe actual voices, such as that he hears people plotting against him. With this there is a rapid progressive weakness, and the patient may fall into dementia and death result from failure of the nerve-centres or from an intercurrent pneumonia, especially

if he has exposed himself greatly. The kidneys may fail and the patient go into a state of uræmia, which latter will be the cause of death.

Prognosis.—This is good as to recovery if the patient's constitution is sufficiently vigorous, but the danger of relapse is always probable. Cases of *mania a potu* should be guarded in prognosis, since the patient may die of a complication, or a permanent dementia may be the sequel. Mild cases usually recover.

Pathology.—There is an alcoholic poisoning of the nerve-centres, producing a chronic congestion of the meninges and the brain, and in the worst cases fatty degeneration of the cortical centres or neurones and the same parenchymatous change in other parts, as the liver and kidneys.

Treatment.—This consists of in the first place withdrawing the drug, which can be done in two ways, (1) suddenly and (2) gradually. The former is the most desired method, immediately stopping the alcohol, supporting the system by means of strychnine or nitroglycerin; and by simple food, such as milk, etc. For the mental excitement, chloral and bromides in combination are indicated. Hyoscine hydrobromate in $\frac{1}{60}$-gr. doses hypodermically is often of great value. The gastro-intestinal tract should be treated carefully; the use of the blue-mass pill, 5 grains, repeated, should be resorted to where the intestinal tract is sluggish. This should be followed by vegetable tonics given before meals. The patient should be kept quiet and exposed to sunlight and fresh air as much as possible. The kidneys should be treated.

Symptoms of the Chronic Form.—These are the same as in the acute form of poisoning, but in less degree. The somatic symptoms predominate; and the mental consist of obtundity, or of dementia in the worst cases. In others the mental symptoms produce the chronic alcoholic insanity or mania, which is a type of excitability or excitable insanity with delusions, and is probably due to a chronic irritation of the cortex from meningeal thickening or patches of cerebral sclerosis.

Diagnosis of any form of alcoholic poisoning will largely depend upon the history of the case and upon the detecting of the drug upon the breath. This, together with the physiognomy of the patient, of acne rosacea, or evidence of congestion of the brain, liver, or kidneys, will make positive the source of the disease when taken with the symptoms detailed.

Prognosis of the Chronic Form.—The chronic form is apt, unless the drug is withdrawn or withheld entirely, to end in fatty degeneration of the brain, heart, etc. This fatty degeneration of the organs of the body and neuritis will occur if the poisoning continues. Chronic alcoholic mania very seldom, if ever, recovers.

Pathology.—Congestion of the meninges of the brain and cord, endarteritis, periarteritis, and sequent fatty degeneration of the brain and various organs of the body, occasionally accompanied by circumscribed hæmorrhages, are all found; as in pachymeningitis hæmorrhagica interna, the symptoms of which are given in another place. Cloudy swelling of the kidneys, the heart, and liver occur; while chronic gastritis and enteritis may complete the pathological picture.

Treatment.—The treatment of the chronic form is largely hygienic and supportive. Alcohol should also be withheld entirely, as in the acute form of poisoning. Nitrate of strychnine is the drug *par excellence*. It is best given hypodermically in $\frac{1}{60}$-grain doses. The uses of cocaine [1] or strong infusion of coffee is of great value as a stimulant in those cases liable to depression after the withdrawal of alcohol. Chronic alcoholic insanity will require treatment in an asylum. Resultant alcoholic neuritis has been treated under another heading. (See Neuritis.)

TOBACCO POISONING

Acute poisoning frequently presents marked symptoms through reflex nervous influence, such as tachycardia, irregular pulse or heart, and palpitation (symptoms that also exist in the so-called tobacco heart in *chronic* poisoning). There may also be disturbances of respiration, mental excitement, and tobacco amblyopia, the latter being common. The poisoning takes place in people who are smokers, or in those who handle tobacco largely, as cigar-makers. The duration of symptoms may be indefinite, and will depend upon the amount of tobacco smoke inhaled and the idiosyncrasy of the patient for or against the drug. The *tobacco heart* is of chronic

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[1] A. J. Hall (Medical News, October 31, 1903) credits caffeine with a physiologic antagonism to alcohol in doses of gr. j or gr. ij every one, two, or three hours. The author contends that in twenty-four or forty-eight hours the drug will effectually quench the craving for alcohol in confirmed *habitués*.

nature, often lasting for many months or several years after complete withdrawal of the poison. Cardiac hypertrophy may occur in it with all the physical signs of heart disease.

Diagnosis.—This is to be made from chronic tea poisoning, which is associated more frequently with chronic dyspepsia, in which latter the amblyopia almost never occurs.

Pathology.—This consists in an auto-intoxication, which may set up fatty degeneration in various organs of the body.

Treatment.—Withdrawal of the tobacco, and supportive measures with nutritious diet are indicated. In tobacco heart it will be necessary to administer vaso-constrictors, as digitalis, strophanthus, etc.; together with rest, fresh air, and sunlight.

TEA AND COFFEE POISONING

Tea and coffee are drugs which very frequently produce symptoms in the nervous system. The symptoms produced are mental excitement, insomnia, tremor, and irregular cardiac action. These are rare, but they should be looked after and attended to in order to prevent bad results. Treatment would be the same as for tobacco poisoning.

LEAD, MERCURY, AND ARSENIC POISONING

A remote common result of these poisons is multiple neuritis.

The symptoms of *lead encephalopathy* are *special* symptoms of chronic lead poisoning, and consist of mental excitement (mania), or general or localized cerebral convulsions, and are usually associated with marked arterial sclerosis set up by the lead. In the pathology of this condition there is also a thickening of the meninges, and later in the course of the disease there is a fatty degeneration of the neurones. The *prognosis* of the disease is bad. The patient may live for many years, however, finally passing into secondary dementia. The treatment should be supportive and eliminative, as potassium iodide for continuous periods. If the mental symptoms predominate, then the same treatment as in insanity should be resorted to.

Mercury and *arsenic* seldom produce nervous symptoms other than the neuritis indicated. The importance of the blue line in chronic lead poisoning and the waxy condition of the skin and the ptyalism produced in mercury are all points which will assist in

making the diagnosis. In arsenic poisoning a point to be taken into consideration is the œdema of the lids (the lower) of the eye. In all of the above poisoning symptoms gastric signs may be expected.

OPIUM POISONING

This manifests itself in the nervous system in the forms of *acute* and *chronic* poisoning.

Symptoms of Acute.—These are slowness of the pulse and respiration; the former may go down to 40 or 50 and the latter to 10 or 12 per minute. The patient becomes comatose, the lips and extremities may become cold, and especially the former blue; besides, the patient may be breathing in a stertorous manner; the secretions of the body are checked, and the pupils become contracted or " pin-pointed." The onset is rather slow and follows some hours after the ingestion of the poisonous drug. Even in the milder cases it is difficult to arouse the patient. While he may appreciate his danger, yet the desire for sleep is so irresistible that he will not make any effort to remain awake even where there is no intended suicide. One grain of sulphate of morphine may produce these symptoms, or from 3 to 10 grains of powdered opium, or smoking of the drug, as a habit. In some cases from the retention of urine there is soon set up a cystic irritation.

Symptoms of Chronic.—The evidence of chronic opium poisoning on the nervous system is shown in mental hebetude, vacillation, etc. The patient is inattentive, generally manifesting to a marked degree a condition of *ego*; but is indecisive, and also cannot, as a rule, advance into new fields of work, similar to that condition existing in paresis. Such a patient's secretions and excretions become scanty, the skin becomes dry and hard, and the physiognomy loses animation, the pupils becoming and remaining contracted. The patient may sit for hours in a " dopy " condition, again at times being vivacious, especially after taking another portion of the drug. It is usually, in most of the cases, seen to be the result of hypodermic medication, the abuse of that form of therapeusis. In other cases smoking of the drug is the cause of the chronic opium poisoning.

Prognosis.—This is rather dubious as to recovery in all cases, since the patient's condition is usually so corrupted at the time he reaches this stage that he is unable to resist the drug which he has

been taking. If this can be reduced, however, liability to cure is proportionately large. The patients may live for years as chronic morphine *habitués*. They are liable at any time to attacks of congestions and low-grade inflammations.

Treatment.—This consists in withdrawing the drug rapidly and completely, and in addition supportive measures should be resorted to. In some cases mental therapeutics, as through hypnotism, suggesting to the patient while in a hypnotic state that he will not be further able to enjoy the use of the drug, will sometimes be of avail. Hyoscine hydrobromate is the only drug that may act as a specific. It should be given in full dosage.

COCAINE HABIT

The abuse of cocaine hydrochlorate has become frequent in recent years. Often the *habitué* has taken the drug as a substitute for opium or some of its alkaloids. Or, again, the habit may have been formed through a patient having taken the drug locally for relief of pain, as in cases of chronic neuralgia of the branches of the fifth nerve, particularly if associated with chronic disease of a tooth or the alveolar processes of the maxillary bones. In other cases the habit has been induced through cocaine sprays in chronic naso-pharyngeal disease. The drug is usually taken by applying pledgets of cotton soaked with 4 per cent or stronger solution to the nasal mucous membrane, or hypodermically.

Symptoms.—The cocaine *habitué* usually shows evidence of mental excitement, associated with cardiac acceleration or irregularity. The respirations are hurried, more pronouncedly after he has recently taken a dose of the drug; when also marked mydriasis, wakefulness, and a sense of diminution of fatigue and hunger. The patient may complain of a sense of "tightness" about the chest. The mucous membranes are dry. In advanced cases the skin is cold and clammy. Other symptoms are impairment of co-ordination, hallucinations, and delirium. Though decidedly diuretic, it lessens the quantity of urea by checking the processes of waste, thus acting as an indirect nutrient, and enabling the body to maintain its energy on a lessened supply of food. On account of the increase of cutaneous circulation local flushings may occur. From the fact that the *habitué* usually tries to conceal the habit, observation as to the small amounts of food taken is important in *diagnosis*.

Prognosis.—The cocaine " fiend " is about as difficult to suc-
cessfully treat as the morphia *habitué.* Permanent cure is always
dubious.

Treatment.—An overdose of cocaine must be combated by
amyl nitrite for the cardiac depression, then alcohol and opium
as stimulants to the heart; also artificial respiration. Chloral and
chloroform are the most directly antagonistic.

The " habit " is best combated by securing full control of the
patient, such as placing him on rest treatment. The drug must
be gradually reduced, an occasional dose of morphia given in
solution that the patient may not detect the substitute. Digi-
talis in 5-minim doses thrice daily or oftener may be required to
tone up the circulation. During the period of withdrawal of the
cocaine the bromides are often valuable to allay cerebral excita-
tion and produce sleep. The gradual increase of proteid food, as
eggs and red meats, is important as metabolism recovers.

COAL-TAR PRODUCTS INTOXICATION

Under this heading come antipyrine, antifebrine, and phenace-
tine as true coal-tar derivatives, while the salicylates are more
strictly of the carbolic-acid series. All of them, when used in
excess in gradually increasing doses, may be said to exist as a
" drug habit."

Symptoms of Poisoning.—As these drugs are used as valuable
remedies for pain, the habit is often engendered very insidiously.
Especially is this so in the hysterical " remembrance pain " follow-
ing true pain of the various painful affections in nervous disease.

There is usually no attempt at deception on the part of the
patient the subject of the coal-tar intoxications, but frequently he
takes the drug with a feeling of its necessity for the real or " re-
membrance pain " referred to above, and to which I have called the
attention of the profession in another place.[1] In toxic doses (in
some cases as much as 100 grains a day of antipyrine in tablets
has been the limit of tolerance) the patient becomes cyanotic,
presenting a leaky skin, feeble, running pulse, singing in the ears,
and a reduction of body temperature $\frac{1}{10}°$ to $\frac{1}{5}°$ F. Occasionally the

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[1] The Therapeutic Status of the Coal-Tar Products in Central Nervous
Affections.   Symposium of the Phila. County Medical Society, Dec. 10, 1902.
Vol. IV, New Series.

patient is nauseated and vomits. Very rarely there is a peculiar erythematous skin eruption noticed over the face, chest, and on the sensitive skin between the fingers. There is a depressant influence upon the brain. In extremely toxic doses the principal influence is exerted upon the blood, altering the shape of the red corpuscles, separating the hematin, and causing decomposition of that fluid.

Diagnosis.—Confusion cannot be made with any other intoxication if the above symptoms are carefully observed.

Prognosis.—This is, as a rule, good in chronic poisoning, since the system has become somewhat immune to the poison.

Treatment.—Of acute intoxication consists in the administration of strong coffee, strychnine, and digitalis, with quietude and alcoholic stimulation. In chronic poisoning, the withdrawal of the drug gradually, the steadying of the circulation by digitalis or strophanthus, quietude (rest in bed), and full feeding are all essentials.

PARESIS

GENERAL PARALYSIS OF THE INSANE—GENERAL PARESIS—DEMENTIA PARALYTICA

THIS is a progressive degenerative disease of the brain, distinguished by abnormal mental symptoms which end in dementia, associated with physical weakness or palsy; also with other characteristic physical signs and symptoms. It runs a fatal course, of about three years' duration in the vast majority of cases.

Ætiology.—Paresis, as it is usually termed, is a parasyphilitic disease, or, as Krafft-Ebing metamorphorically puts it, a disease of syphilization—in consonance with another fact that it is a disease of modern civilization. A century ago paralytic dementia was rare, now it is seen frequently at clinics, asylums, and in private practise. The disease is rare still in Asia and Africa. Climate in these countries must have some immunizing influence upon the system, since the same cases may be as likely to acquire paresis if exposed to specific infection when in Europe or America. It is a disease of indirect heredity to predisposition—i. e., the patient is born with a neuropathic constitution, though it *is* once in a while found to be congenital, occurring in the very young whose parents were syphilitic. It occurs almost six times as often in the male as in the female. As women take on the follies of men, we find the proportion of cases increasing in the female sex.

Excessive mental exertion, as of the intense mind tension of a banker, is a predisposing cause also, as is alcoholism. Sexual excess may deplete the central nervous system neurones to such an extent as to greatly predispose to paresis, but is almost never an *exciting* cause, as has been thought. This disease, with locomotor ataxia, must be put down as being in 75 per cent of cases (more in our experience) due to syphilis. Krafft-Ebing gives 80 per cent as being specific; and when occurring in children Zappert makes

the proportion of cases of paresis due to syphilis over 87 per cent. Sunstroke, exposure, injuries, and acute diseases are also exciting causes in the minority of cases. But once having had syphilis associated with any other of the causes enumerated, such a one is very likely to develop paresis in from eight to fifteen years after the initial lesion. The disease occurs more frequently between the ages of twenty and forty, most usual about the thirtieth year. It is more frequent in married people, and is found oftener in city than country, for obvious reasons already inferred.

Symptoms.—There are *two* stages: one of *excitement* or irritation, the other of *dementia* and paralysis. Substitution for the first stage may be in a prolonged period of melancholia or hypochondriasis. In other cases the dementia and bodily enfeeblement may go hand in hand from the first. The *first* type is the most common, although the opinion is growing that mental disturbance is becoming less in importance than is the initial physical weakness. Thus it is assumed that paresis is becoming much more a disease of the brain and cord and less a disease of the mind, the mental symptoms being that of a progressive dementia from the incipiency of the process.

Usual Type.—*First stage:* The patient shows unusual irritability of temper; he is annoyed by trifles, his family noting the marked change in disposition to the household; also a lack of proper appreciation of business relations. He becomes peevish, is easily fatigued, is exhausted without cause, and is unable to fix his attention for any length of time upon his affairs. He is apt to be ex-

Fig. 79.—Paretic Dementia. Ordinary Type, Second Stage. (Philadelphia Hospital, Insane Department.)

travagant or make some foolish error in judgment. This condition of irritability is followed by great exaltation. He becomes

happy, and possesses a feeling of " well-being." He is effusive of speech, and jocose where he was formerly sober or reserved. He develops great ideas, as of money-making, and all by a conglomerate mass of ideas, such as by a meat-market and automobile establishment combined. These delusions of grandeur (megalomania) are more or less periodic, and the period of exaltation often gives place to outbursts of violence, this latter being more pronounced if he has developed a transient dipsomania, which is not unusual. Within a few months the family usually solicit aid of the physician, and the patient is confined in an institution where he can do himself or others no harm. Often under this restraining influence *apparent* remissions occur, and occasionally *real* remissions occur, as in a case of the writer's where ten years had elapsed since a staying of all symptoms of undoubted paresis has occurred, the man still holding the initial symptoms.

Usually, however, after some months in a hospital the patient begins to show marked signs of dementia, memory becomes worse, he misplaces objects, forgets recent matters, commits errors in figuring, cannot write coherently, or may make mistakes in spelling or elision of letters. The handwriting becomes wavering, the letters showing irregularities in the strokes.

During the exaltation period physical signs develop which are characteristic. Besides the difficulty in writing, noted above, there is decided facial tremor, brought out the more when the patient is told to close his eyes and stretch the muscles of the face so as to show the teeth. Marked tremor of the tongue is present, much like that of acute alcoholism. Speech becomes thick; he stutters; it is very difficult to pronounce long words. These evidences of dysarthria are entirely cerebral in origin. The knee-jerks are usually increased. The pupils are generally unequal; they are apt to react badly to light,[1] and even sluggishly to accommodation. The fundi oculi are normal.

An early and decided weakness of the sexual function is found. The vesical sphincter may become paretic, allowing " dribbling " of urine. Appetite may be increased, manner of taking food gluttonous. Vegetative organs remain normal until very late in the

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[1] The true Argyll-Robertson pupil, recorded in the books, the writer has not found in twenty-five out of one hundred cases carefully examined. Irregularity and iridoplegia he would beg to submit as being the most characteristic pupillary signs in paresis.

course of the disease. The patient often suffers from insomnia, or he may be subject to vertiginous, syncopal, or pseudo-apoplectiform attacks, in the latter of which he may fall over and be a sufferer from hemiplegia, lasting for a few days or even weeks. Epileptic convulsions may occur. In rare cases there is abolition of the knee-jerks and ataxic symptoms somewhat resembling the paretic stage of tabes. In such cases the general muscular power is much diminished, the patient being unable to do any sustained muscular work, as walking any distance. *True* apoplexy may occur.

In the *second stage* the most striking sign is the gradual onset of dementia. The patient sleeps much in daytime and becomes more quiet. He is very forgetful now, may not at times even recognise his friends, and, of course, takes little interest in outside affairs. He cannot speak of current events, forgets the day of the week, etc. He soils himself at meals, slobbers his liquids, and takes no interest in his personal appearance. He may be uncleanly at the toilet, and finally becomes as dependent as a small child. Later apoplectiform attacks occur with permanent hemiplegia, as noted above. He may now gain flesh and have a very voracious appetite. In periods of excitement at this stage he may have delusions of persecution or slight delusions of grandeur. Tremor increases and speech becomes distinctly worse. The average duration of the disease is about three years. Some may die within a year, or remission may occur and the patient live in dementia fifteen years in rare cases. [A patient of mine imagined he gained untold wealth from day to day, although he did not name any specific sum. He then quickly became bedridden and helpless.]

Dementing Type.—In this form there is a primary dementia, beginning without any excitement. First there is lack of attention to business, forgetfulness, and general incapacity for work. The patient makes mistakes in dates, in calculations; is good-natured to everybody about him, is passively happy. *He may have no delusions of any sort.* Somatic symptoms of tremor, fixed pupils, exaggerated reflexes, finally appear and become characteristic. The scanning speech may appear last of all in this type. There may be increase of phosphates in the urine, particularly showing during any excitable period. Excess of uric acid we have also found in these cases. Leucocytosis is generally present. The red blood-cells become qualitatively changed, there being a relative

increase of the polynuclear cells, very much as shown in a study
with Dr. Napoleon Boston [1] to exist in idiopathic epilepsy.

Hypochondriacal Type.—In this form the symptoms begin with
those of hypochondriasis and neurasthenia. The patient complains
of a vague feeling of distress about the head, of hemicrania at
times; also of pain in the back and limbs. One of my recent
cases complained most distressingly of dull pain in the epigastrium,
the tongue being constantly coated, and large amounts of indican
were found in the urine. Vague sense of discomfort also occurs,
which the patient at his best (in the early morning) is still unable
to describe. Frequently these are treated for weeks as cases of
neurasthenia, since at the start they scarcely show any physical
or mental symptoms. But careful study of anamnesis will reveal
in the history syphilitic infection, while a study of the physical
signs will show the rigid pupils as perhaps the only clue to somatic
failure. After a year or so mental unbalance appears, shown in
suspicion, dementia, delusions of persecution, and the like. Occa-
sional outbreaks of violence may occur, then all the typical signs—
alteration of speech, tremor, etc.—will follow.

Syphilitic Pseudo-Paresis.—In some cases of paresis symptoms
of exudative syphilis introduce the disease. The patient has at
first palsies of the eyes, or attacks of hemiplegia with intense head-
ache, followed by convulsions. The specific exudate is found to
either be pressing against the convexity or more usually against
the base. Under proper treatment resolution of the granulomata
occurs and the seizures or paralysis disappear, but it is now found
he is emotional, is excitable, his memory is impaired, judgment is
weakened; he has to live a quiet, inactive life, giving up his regu-
lar employment or professional work. A pronounced dementia
may not set in for eight or nine years in this class of cases.

Alcoholic Pseudo-Paresis.—Those who have for a long time
continuously indulged in alcohol or who have been debauchees in
abuse of alcohol may finally develop—usually do—a condition of
mental weakness simulating paresis. I had a patient of emi-
nently nervous temperament, but of good ancestry, who, follow-
ing troubles in his family, drank, not so heavily, but periodically,
to the production of *mania a potu* on several occasions only. This
was followed by a typical form of pseudo-paresis due to acute
alcoholic intoxication.

[1] Transactions of the College of Physicians of Philadelphia, April 1, 1903.

In these patients, there having been no syphilitic infection, there is no true paresis. Such patients are selfish, at times become bestial, while at all times they show feeble judgment; the memory becomes a blank. There is a gradual loss of the moral sense. Dementia progresses slowly, but these cases are usually carried off by some intercurrent disease. They never present the physical signs nor typical symptoms of true paresis. If alcohol is withheld they may live many years.

Pathology.—Paresis is a parasyphilitic disease (90 per cent) due to degeneration of the cerebral neurones from the syphilitic toxines developed late in the luetic malady. There is first thickening of blood-vessels due to periarteritis, endarteritis, and proliferation into the perivascular tissue. It is in doubt whether the degeneration of the nerve-cell is really secondary to the above changes; or is primary, as is the case in tabes. Most authorities cling to the latter view, which is probably the correct one.

On examination, the brain shows thickening and vascularization of the dura mater. This membrane is generally partially adherent to the cranial bones. The brain mass itself has usually undergone atrophy, as shown by decrease of a few ounces in weight over the normal. There is also increase of cerebro-spinal fluid. The pia mater in the fore and mid regions of the brain is much congested. It and the arachnoid are much thickened. The latter is adherent to the brain throughout, and when torn away the cortex is found to be œdematous.

Microscopic study reveals increase of the connective tissue and the neuroglia about the blood-vessels of the cortex, thickening of the interna and all the vascular coats to some degree, together with degeneration of nerve-cells at all ages. Not only the neurones of the gray cortex, but those of the medulla, are also involved in the fatty change.

Lesions of the posterior and lateral columns of the cord may also be found in a third of the cases. Therefore locomotor ataxic symptoms will be found in $33\frac{1}{3}$ per cent of cases of paresis. (A less number of cases of primary ataxia (4 per cent) will end in paresis—the third stage of ataxia. (See Tabes.) This is due to the fact that tabetics usually are carried off early by other disease.)

Prognosis.—This is invariably unfavourable. The patient may often, however, be partially restored by the use of tonics, gen-

eral remedial and hygienic measures, which includes with particularity the removal of the patient from all forms of excitement; also the enforcement of quietude. The use of specific medication in acute early developing cases after the luetic infection may also tend to stay the progress of the disease. Although the use of mercurials and iodides will not cure or stay the advanced stage, I have seen in the early stage great good result from their use; in several cases apparent cures result. When the frank typical symptoms once develop, cure is impossible at the present stage of our art. Remissions and the milder type of the affection within the last decade have a more promising hope for greater accuracy in scientific treatment, awaiting only future development.

Treatment.—In the early developing cases, as indicated, place the patient promptly on antisyphilitic treatment—K. I., gr. v, t. i. d., $HgCl_2$, gr. $\frac{1}{48}$, t. i. d., or even the use of inunctions of mercurial ointment, a dram daily. The patient should be kept absolutely under control by removing him to some hospital or institution for mental and physical quiet, with an abundance of fresh air and sunlight about him. Tonic measures, such as the use of the Brown-Séquard testicular mixture (in glycerine), I have seen do good in stimulating the patient and aiding metabolism generally. The use of the ductless glands, as thyreoid or adrenals, may also, by aiding nutrition and oxidation, be of service in staying the disease. Glycero-phosphate of lime, 10 grains three times a day, I have seen give much benefit. The Charcot douche, cold sponge daily, and light massage aid much in procuring better circulation. Bromides must be employed as periods of excitement indicate. Hyoscine hydrobromate, gr. $\frac{1}{60}$, hypodermically, is of great value in maniacal outbursts. Chloral and morphia should be cautiously employed.

APPENDIX

DISORDERS OF SLEEP

CONSIDERATION of the physiology of sleep and waking has been studied in the forepart of this work (Chapter I, pp. 42 and 43).

The symptomatic disorder, insomnia, has also been treated at length in another place (p. 126).

Sleep is that calm to the nervous system that supplies nerve energies for the workingman. There are many theories of the causation of sleep, the latest being that of retraction of the protoplasm of the cortical neurones. This separating one from another cuts off the association of nerve impulses of the higher centres, while those lower more physical or vegetative functions still are active. This beautiful mechanism is thus well illustrative of the possible gross mechanism of mind action.[1] It at least permits of better conception of the ever mysterious phenomena of sleep. The causation of sleep is more universally, however, considered to be due to anæmia of the brain. This theory has its partial proof in the fact that the blood pressure is lowered during sleep, as shown by sphygmographic tracings. Self-developed catabolites may be the remote cause back of all others; these autochthonous poisons precipitating the mechanical status of the cells found during sleep.

Given a healthy mind in a healthy body, we have the highest type of man: *mens conscia recti.* So that the wear and tear of life is constantly balanced in part by periods of repose, particularly of sleep. There is never an absolute repose of mind or body, however, from birth to death.

There is nothing so subtle or important as the study of the disorders of sleep. In the first place, we must recall that there is

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[1] See the discharge theory suggested by Bechterew (Neurol. Centralbl., 1896, Nos. 2 and 3); also the studies of Forel, His, and Cajal abroad, and Dercum in this country (Functions of the Neurone, Jour. of N. and M. Dis., August, 1896).

an individuality about the amount of sleep necessary as a restora-
tive agent. Some nerve protoplasm requires more hours of sleep
than another. Nor is it the neurotic person that requires most.
It is simply a matter of idiosyncrasy, which only can be judged
by the history of any case. In proportion as sleep does service in
resting the mind, do we have the only accurate index of the value
of a given slumber; and yet, as some one has well said, a dreamless
sleep probably does not occur. We should reason from this that
fixing or a resetting of our thoughts during the time of peaceful,
quiet sleep is one of its functions. Indeed, too, the mental impres-
sions thus finally fixed during sleep are probably of the most en-
during nature—the mainsprings of character; for consciousness
brings its artificialities, while the various pitfalls of convention-
ality are entirely done away with in sound sleep, and the best
things of the mind remain afterward. It is but the demonstration
of God's rule as set down by Darwin, " the survival of the fittest."
It is a high province of the physician to bring about natural sleep.

A. WAKEFUL DISORDERS OF SLEEP

Insomnia may be said to be the most important disturbance of
sleep. Its causes are legion: pain, lack or excess of exercise,
toxæmias, excitement, or worriment. In the treatment of it no
physician can be successful unless he seeks out the fundamental
cause.

Pain, if intractable, must be counteracted by a dose of mor-
phine. Poisons should be eliminated. Exercise must be regulated
to oxygenate the blood properly; a happy medium for the indi-
vidual case is to be sought. It is folly to advise long walks for the
banker or a series of rigid calisthenics for an overworked lawyer or
physician or student. This I have seen do almost irreparable dam-
age in a neurasthenic man attending the regular gymnastic course
in one of the leading universities of New York State. Excite-
ment must be *ad minimum* in the sufferer from insomnia. It is
well also to arrange, for the several hours preceding " bedtime,"
especial mental and physical quietude. The room should be well
ventilated in which the patient sleeps. It should be dry, prefer-
ably in a room of northern exposure, since many are easily wak-
ened, as by rays of the rising sun at daybreak. All extrinsic causes,
such as noises, should be guarded against. I have seen patients

immediately improve by being taken to another room when some particular, though slight, noise, not thought of by the sufferer, was the cause of his awakening.

General methods to employ are warm water bathing (temperature 112° F.), followed by a brisk rub with a Turkish towel; or light massage, especially stroking the forehead at the end of the treatment; or hot milk taken at the moment of retiring. Hypnotism may be resorted to in the exceptional case where hysteria is the base of the trouble, but it is not a reliable agent, and is liable to abuse.

Where high arterial brain pressure exists the cautious employment of depressants, as aconitia, gr. $\frac{1}{50}$ at bedtime, or several minims of tincture of aconite, I have seen of positive service. But any attempts to relieve vascular pressure by nitro-glycerine will aggravate the case, due to increasing the flow of blood to the surface, and therefore the meninges, thus causing irritation of the branches of the fifth nerve.

Somnambulism is sleep-walking, a state in which the patient acts his part of a dream. In this disorder the subject passes, more or less automatically, through various apparently natural phases of physical and organic life, without full mental inhibition existing as when the *conscious* mind controls him. Somnambulism is, to a degree, evidence of unrest, of inevitable wakefulness. I have a patient who walks about at night frequently, talks coherently the while to those who address her, but in the morning she is exhausted and remembers nothing of the phenomena. Thus, too, it is with people who dream constantly. It is evidence that the physiological mechanism is out of kilter, so to speak, especially if the dream is something "horrible" or unpleasant. "Pleasant dreams" are, however, natural and not in any extent exhausting, because they *are* natural, and any natural event is a stimulation of metabolism without which latter mind action cannot be at its best. The proverbial saying that chronic invalids develop frequently the best mental and moral specimens of men and women is not borne out by the cold facts, as physicians so well know. They are good specimens in *spite* of their infirmity only. Disorders of digestion have much to do with both of the above states. One so disposed should be instructed to be very cautious about indiscretion in diet. An hypnotic given at bedtime, preferably trional, grs. xv, or chloralamid, gr. xij, will aid to prevent

attacks of either. The hygienic care is, in other respects, the same as in simple essential insomnia, a most important matter.

B. Somnolent Disorders of Sleep

These are the disorders marked by an irresistible desire to sleep, and they are not so frequent as Class A, but usually imply the same grave organic disease or incurable affection, where the underlying pathology is not as yet definitely determined, except in hysteria, which may simulate any malady.

Ecstasy, catalepsy, and trance are accidental somnolent states occurring in hysteria. Ecstasy is a more or less transient emotional state when the patient is transfixed, as it were, in self-contemplation. When there is general muscular rigidity associated for some period of time, catalepsy exists. A trance is a passive condition of quiet somnolence in which the patient lies motionless with all function at the lowest ebb. Respiration and pulse may be almost imperceptible.

The treatment of these three conditions will be that of the management of a general hysterical state. It should be mentioned that some complex treatment, such as hypnotism, or a long series of regulated exercises in deep breathing, the mental and hydrotherapeutic value of a cold shower-bath, together with forced feeding to the constitutional limit, singly or combined, will offer the best rewards in cure to the caretaking physician.

Sleeping sickness, as recently described by Manton and Mott as due to filaria and occurring in certain natives of Africa, is a clinical syndrome in which somnolence appears in intervals gradually increasing until the patient sleeps steadily for many hours. At first the patient can be aroused sufficiently to take food, and finally awakened with extreme difficulty.

In two cases studied by Mott [1] a chronic infused leptomeningitis and encephalomyelitis were found, but no infectious organism. In studying the blood [2] for another purpose with Dr. Napoleon Boston, this observer discovered in a rat's blood an

[1] British Medical Journal, December, 1899.
[2] The Blood in Epilepsy: Experiments on Animals. Transactions of the Physicians of the College of Philadelphia, March, 1903. American Journal of Insanity, January, 1904.

actively motile micro-organism similar to the one described by Manton very recently as a probable *cause* of sleeping sickness. Upon a warm slide, with the lower power of the microscope, the organisms could be readily seen pushing the corpuscles about. The organism has been designated *Tryfanosomos Castellani* from its discoverer.

If this organism is the cause of this disease some form of immunization or prophylaxis seems to be the only possible hope for cure in the future.

Narcolepsy is a condition in which the patient repeatedly goes to sleep in the day. It is a marked form of epilepsy and requires the same treatment (see Epilepsy).

ADDITIONAL FIGURES

FIG. 80.—SPASTIC AND ATHETOID MOVEMENTS IN FACE, TONGUE, AND PHARYNGEAL MUSCLES. CASE OF CEREBRAL SPASTIC PARALYSIS. (Philadelphia Hospital.)

FIG. 81.—ATTITUDE IN A CASE OF GENERAL ATHETOSIS IN CEREBRAL PALSY OF
CHILDHOOD.   (Philadelphia Hospital.)

FIG. 82.—SECONDARY CONTRACTIONS OF FACE, ARM, HAND, FOOT, AND LEG IN CASE OF CHRONIC LEFT HEMIPLEGIA. (Philadelphia Hospital.)

FIG. 83.—TUMOUR (ENDOTHELIOMA). INNER SURFACE CEREBRAL DURA. (Museum of Philadelphia Hospital.)
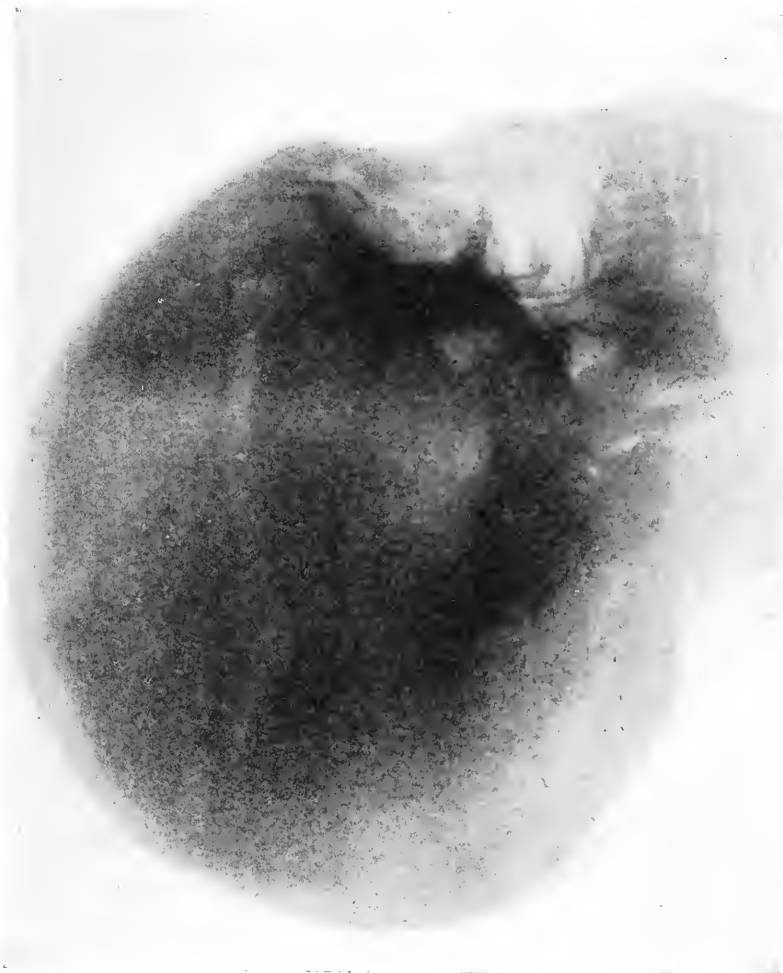
Fig. 84.—Horizontal Linear Light Shadow (Radiograph), showing Result of Specific Meningitis and Erosion of Inner Plate of Parieto-Frontal Bones. (Kindness of Dr. Boggs, Pittsburg, Pa.)

Region of the central convolutions.
Region of the third frontal convolution and the insula (speech center).
Temporalis.
Facial (upper) in front of ear.
Facial (trunk).
Posterior auricular.
Facial (middle br'ch).
Facial (lower branch).
Splenius.
Sterno-cleido-mastoid.
Spinal accessory.
Levator anguli scapulæ.
Trapezius.
Dorsalis scapulæ.
Axillary (circumflex).
Long thoracic (serratus magnus).

Brachial plexus.

Supraclavicular point. (Erb's point. Deltoid, biceps, brachialis anticus, and supinator longus.)

Phrenic.

Frontalis.
Facial (upper branch).
Corrugator supercilii.
Orbicul.palpebrarum.
Nasal muscles.
Zygomatici.
Orbicularis oris.
Facial (middle br'ch).
Masseter.
Levator menti.
Quadratus menti.
Triangularis menti.
Hypoglossus.
Facial (lower branch).
Platysma myoides.
Hyoid muscles.
Omohyoid.
Anterior thoracic (Pectoralis major).

Fig. 85.—Showing Motor Points. (Head and Neck.)
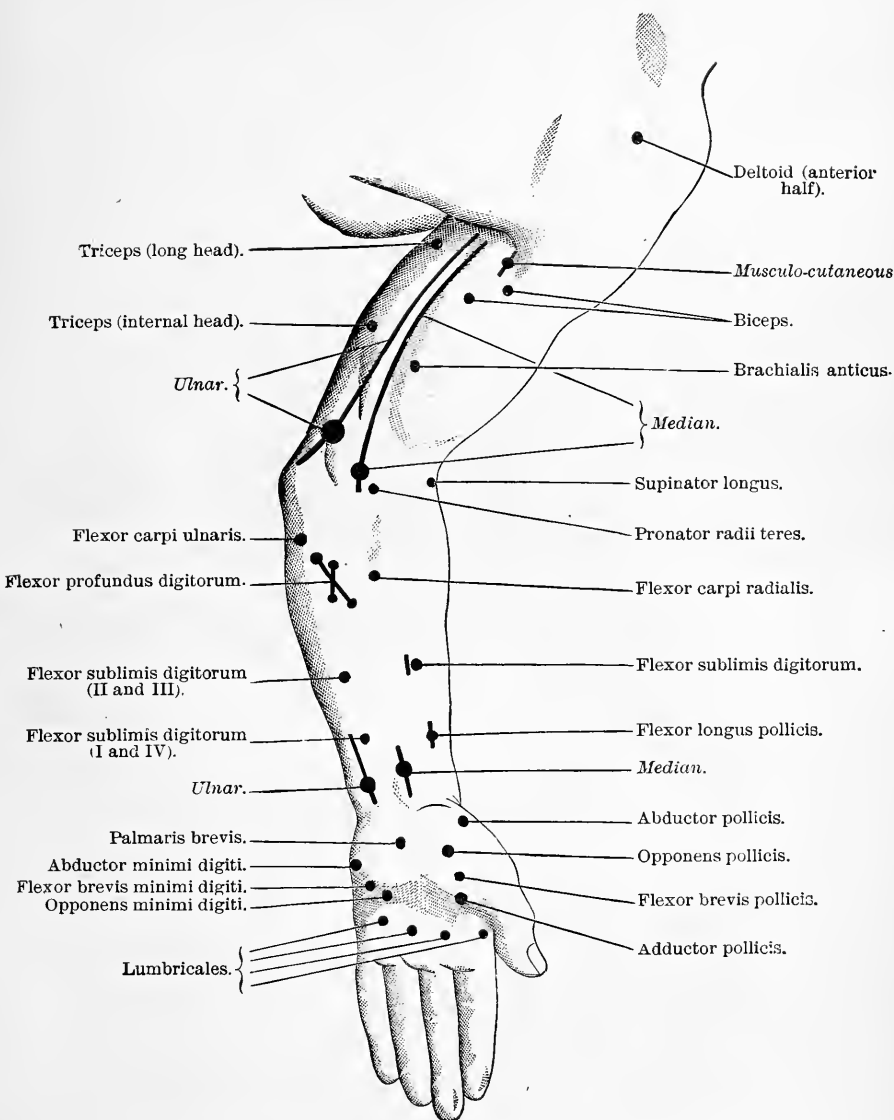
FIG. 86.—SHOWING MOTOR POINTS. (Shoulder and Arm.) Anterior Aspect.
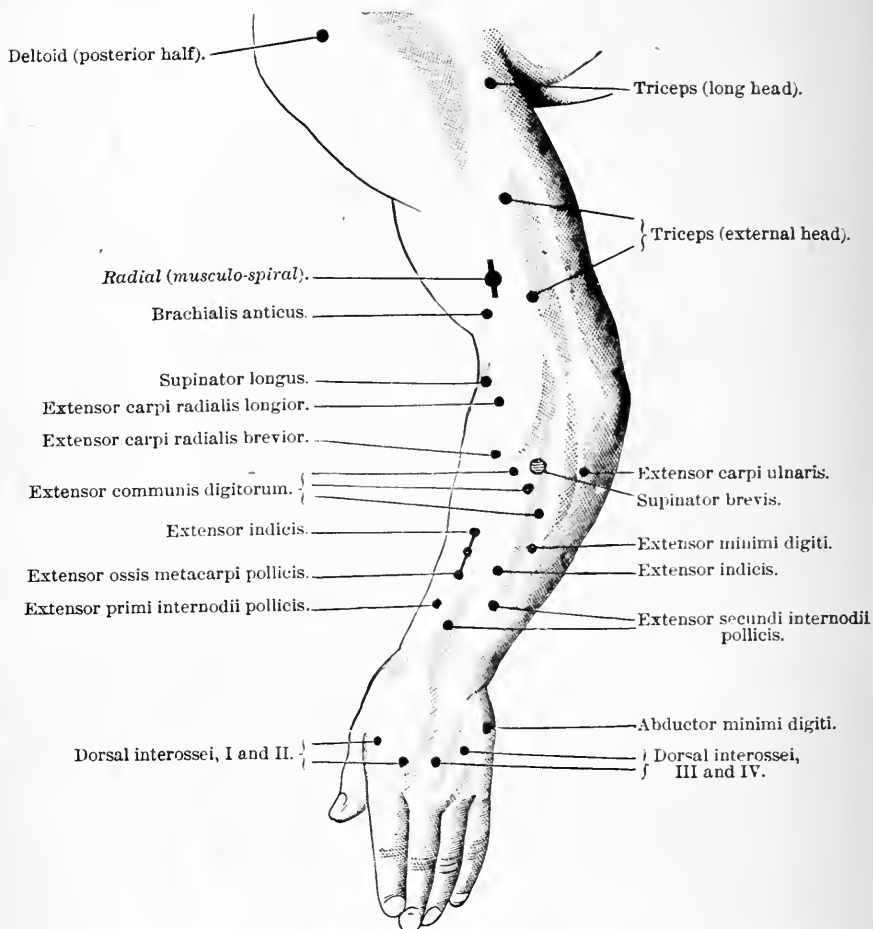
FIG. 87.—Showing Motor Points. (Shoulder and Arm.) Posterior Aspect.

Peroneal.

Gastrocnemius (external).

Tibialis anticus.

Peroneus longus.

Extensor longus digitorum.

Peroneus brevis.

Soleus.

Extensor proprius pollicis.

Flexor longus hallucis.

Extensor brevis digitorum

Dorsal interossei. {

Abductor minimi digiti.
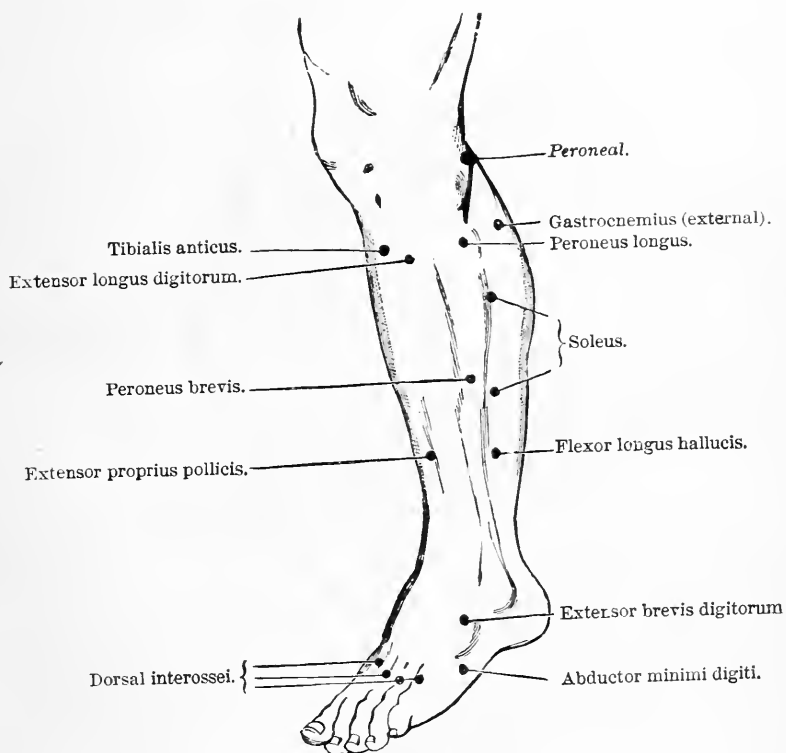
FIG. 88.—SHOWING MOTOR POINTS.   (Leg.)
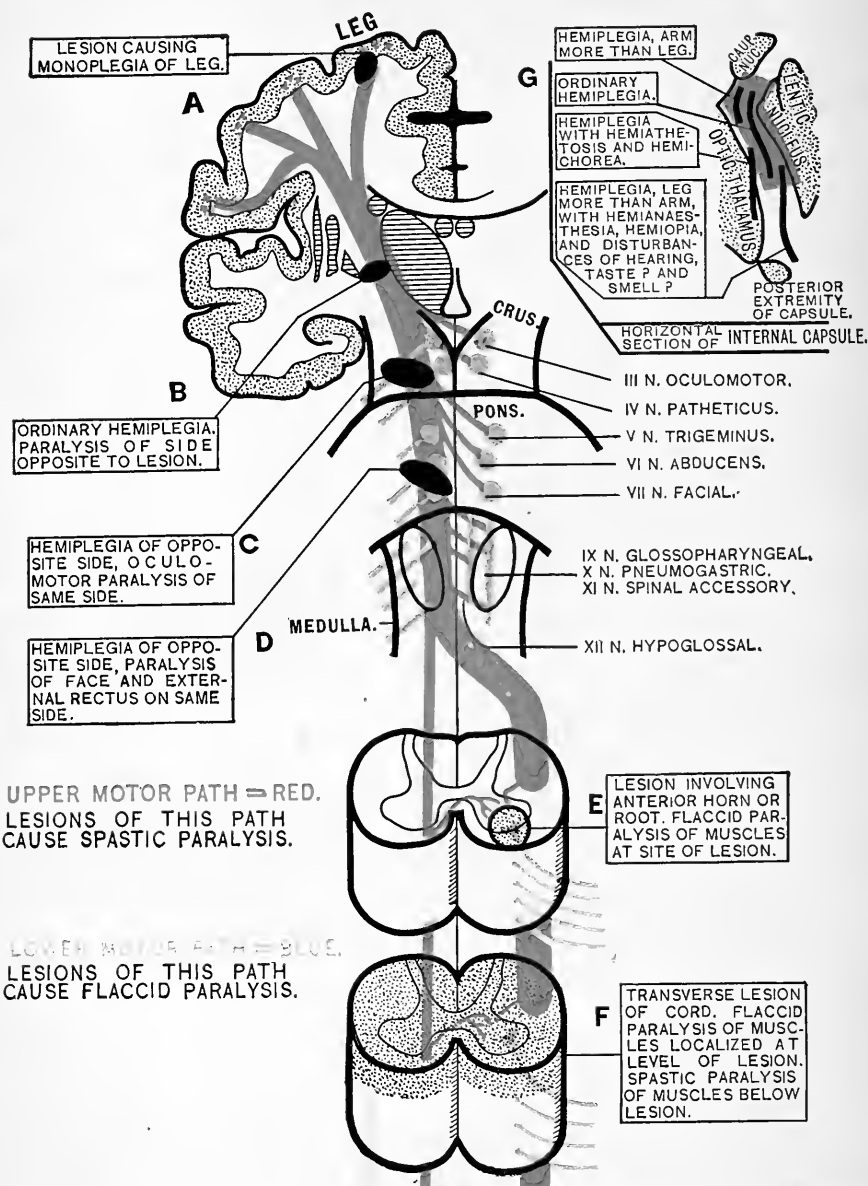
FIG. 89.—SHOWING THE EFFECTS OF VARIOUS LESIONS OF THE MOTOR PATH IN THE BRAIN AND SPINAL CORD. THE UPPER RIGHT-HAND SKETCH SHOWS THE VARIATIONS IN SYMPTOMS CAUSED BY A DIFFERENCE IN THE ANTERO-POSTERIOR POSITION OF THE LESIONS IN THE INTERNAL CAPSULE.
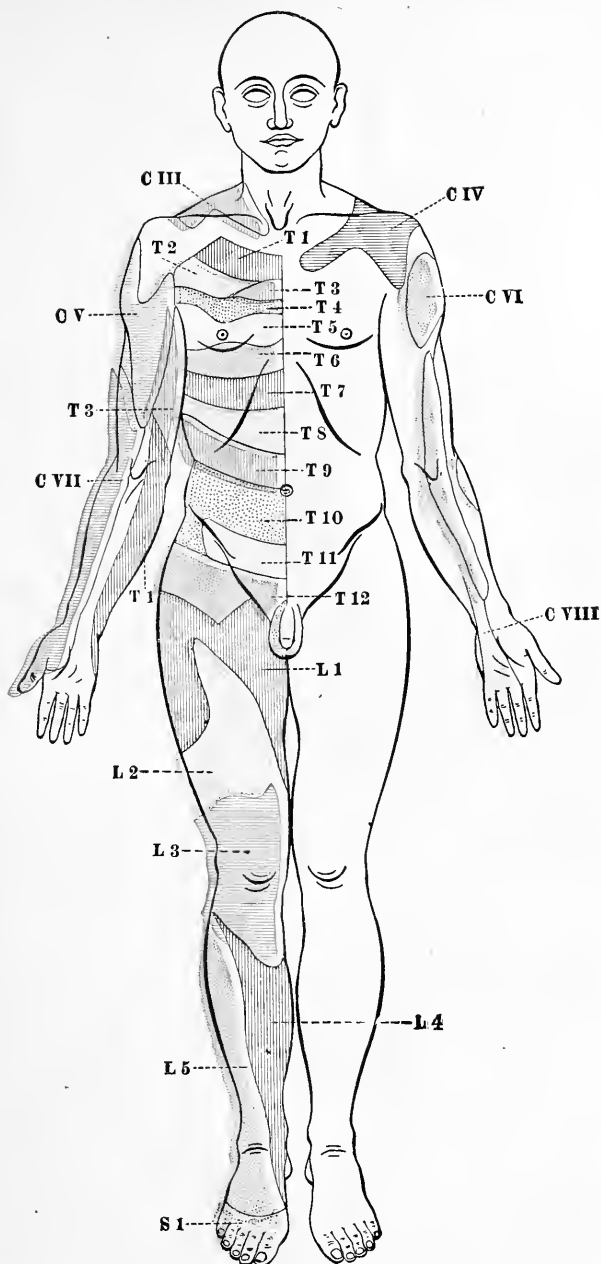
FIG. 90.—DIAGRAM OF SKIN AREAS CORRESPONDING TO THE DIFFERENT SPINAL
SEGMENTS. Anterior Aspect. (Combined from Head's Diagrams by Osler.)
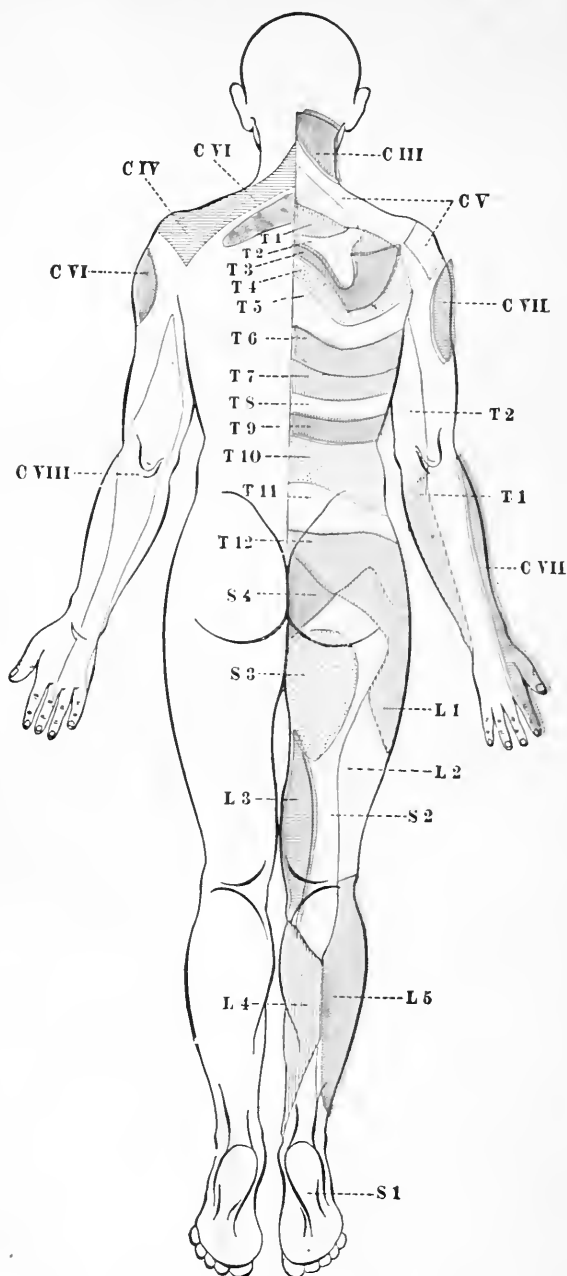
FIG. 91.—DIAGRAM OF SKIN AREAS CORRESPONDING TO THE DIFFERENT SPINAL SEGMENTS. Posterior Aspect. (Combined from Head's Diagrams by Osler.)

Tables

The difference between multiple neuritis and poliomyelitis is well shown in the following modification of the summary by Starr:

Acute poliomyelitis	*Multiple neuritis*
Sudden onset, with fever and development of paralysis in all the limbs, followed in from three to five days by subsidence of paralysis, which remains in a few muscles of one limb; or, if two are affected, the paralysis is rarely symmetrical. If the onset is subacute, four weeks is the duration of onset. The muscles are not tender. Sensory symptoms are rare, and, when present, soon subside.	Fatigue for some weeks; then sudden onset and progress for two weeks with or without fever. Legs usually first affected, then arms, then body. The paralysis has no tendency to subside for some time (months). The limbs are affected symmetrically. Usually there is tenderness of the nerve-trunks and affected muscles, and pain, with areas of anæsthesia.

Gowers's Table of Laryngeal Paralysis

Symptoms	*Signs*	*Lesion*
No voice; no c o u g h ; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy. •
Voice l i t t l e changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor nor dyspnœa.	Cords normal in position and mo‐ing normally in respiration, but not brought together on an attempt at phonation.	Abductor palsy.

The following table from Sachs shows the lesions most likely to be present, according to the time of onset in Cerebral Palsies:

Groups.		Morbid Lesion.
Diaplegia and paraplegia	Paralysis of intra-uterine onset.	Large cerebral defects; porencephaly. Defective development of pyramidal tracts. Agenesis corticalis (highest nerve-elements involved).
	Birth palsies.	Meningeal hæmorrhage, rarely intracerebral hæmorrhage. Later conditions, meningo-encephalitis chronica, sclerosis, cysts, partial atrophies.
Hemiplegia	Acute (acquired) palsies.	Hæmorrhage, meningeal and rarely intracerebral; thrombosis, from syphilitic endarteritis and in marantic conditions ; embolism. Later conditions, atrophy, cysts, and sclerosis (diffuse and lobar). Meningitis chronica. Hydrocephalus (seldom the sole cause), primary encephalitis, polio-encephalitis acuta (Strümpell).

DIFFERENTIAL POINTS OF THE TWO FORMS OF " FAMILY ATAXIA "

(I) *Friedreich's Form*

1. Occurs before puberty.
2. Choreiform movements seen affecting head, arm, and trunk.
3. Optic atrophy exceptional.
4. Tendon reflexes diminished or abolished.
5. Club-foot and scoliosis common.

(II) *Marie's Form*

1. Occurs after puberty.
2. Choreiform movements are *very* pronounced.
3. Optic atrophy is common.
4. Increased reflexes and clonus frequent.
5. Club-foot and scoliosis are exceptional.

FORMULARY

For anæmia in neurasthenia.

 ℞ Pil. Blaud gr. v $\begin{cases} \text{Ferri sulph.} \\ \text{Potass. carbonat.......āā gr. ijss.} \end{cases}$

 Sig.: One or two pills ten minutes after meals.

Neurasthenia (essential).

 ℞ Sod. brom. }

  Ammon. brom. } ....āā ʒ ij.

  Aquæ...........................q. s. ad ℥ iij.

 M. et sig.: Teaspoonful every three hours until head symptoms relieved.

Neurasthenia (insomnia of).

 ℞ Trional.................................ʒ ij.

 M. et ft. chart No. x.

 Sig.: One at nine o'clock in hot milk.

Neurasthenia (sexual).

 ℞ Ferri valerianat

  Zinci valerianat } ....āā gr. xxiv.

  Quin. valerianat

 M. et ft. pil. No. xxiv.

 Sig.: One after meals.

Also neurasthenia (sexual) with irritable bladder.

 ℞ Potass. citratis........................ʒ iij.

  Essence pepsini ....fl. ℥ j.

  Aquæ cinnamonii ....℥ iij.

  M. et sig.: Teaspoonful every three hours.

For nervous cough.

 ℞ Ammon. chloridi.......................ʒ jss.

  Sod. bromidi...........................ʒ iij.

  Tr. opii. camph........................fl. ℥ ss.

  Aq. menth. pip.................q. s. ad fl. ℥ iij.

 M. et sig.: Teaspoonful every three or four hours.

For dyspepsia (nervous) with gastrectasia.

℞   Tr. nucis vom.......................... fl. ʒ j.
    Sodii bromidi......................... ʒ vj.
    Tr. aurantii.......................... fl. ʒ j.
    Tr. gent. comp...................q. s. ad fl. ℥ iij.
M. et sig. : Teaspoonful a half hour before meals.

Also

℞   Ext. chiratta ⎫
    Ext. gentian ⎭ ....āā gr. **xxiv.**
M. et ft. capsule No. xxiv.
Sig. : One a half hour before meals.

Also

℞   Tr. nucis vom........................ fl. ʒ **iv.**
    Sod. bicarb.......................... ʒ jss.
    Inf. gent. comp...............q. s. ad fl. ℥ iij.
M. et sig. : Teaspoonful before meals.

For nervous syphilis.

℞   Hydrarg. chlor. corrosive............... gr. **j.**
    Potass. iodidi........................ fl. ʒ ij.
    Aquæ cinnamonii...............q. s. ad fl. ℥ iij.
M. et sig. : Teaspoonful after meals.

Nervous syphilis (tonic in).

℞   Tr. nucis vom........................ ʒ ij.
    Elix. calisayæ..................q. s. ad ℥ iij.
M. et sig. : Dessertspoonful every three hours.

For hysteria.

℞   Pil. asafœtida........................ gr. **iij.**
Sig. : One three or four times a day.

Hysteria and anæmia.

℞   Pil. sumbul comp. ⎧ Ext. sumbul........ gr. **j.**
                       ⎪ Ferri sulph. exsic.... gr. **j.**
                       ⎨ Asafœtida.......... gr. **ij.**
                       ⎩ Acid. arseniosi...... gr. $\frac{1}{40}$.

M. et ft. pil.
Sig. : One three or four times a day.

Headache (simple).

    ℞   Acetanilid .... gr. ij.

         Camphor monobrom.................... gr. ss.

         Caffeine citratis. .... gr. ss.

    M. et ft. pil. No. i.

    Sig.: One pill every three hours until four doses if required.

Headache (alcoholism).

    ℞   Tr. nucis vom. ⎱

         Tr. caprici   ⎰ .... āā fl. ℥ j.

         Sod. bromidi......................... ℥ ss.

         Tr. gent. comp...................q. s. ad fl. ℥ iij.

    M. et sig.: Teaspoonful every three hours.

For headache (la grippe).

    ℞   Salol   ⎱

         Phenacetine ⎰ .... āā ℥ ss.

    M. et ft. cap. No. xii.

    Sig.: One capsule every two or three hours for three doses.

For headache (cerebral congestion and uricacidæmia).

    ℞   Sod. salicylat......................... ℥ iij.

         Codeine sulph........................ gr. v.

         Elix. calisayæ........................ fl. ℥ j.

         Aquæ...........................q. s. ad fl. ℥ iij.

    M. et sig.: Teaspoonful every four hours.

For mental depression (bilious temperament).

    ℞   Acid. nitrohydrochlor. dil............... fl. ℥ ijss.

         Tr. nucis vom........................ fl. ℥ iij.

         Tr. aurantii......................... q. s.

         Tr. gent. comp...................q. s. ad fl. ℥ vj.

    M. et sig.: Teaspoonful a half hour before meals.

For paræsthesia.

    ℞   Ext. cascara sagrada fld................ fl. ℥ j.

         Ext. ergot fld........................ ℥ ij.

    M. et sig.: Teaspoonful twice daily.

For Stokes-Adams syndrome.

    ℞   Nitroglycerine .... gr. $\frac{1}{200}$.

    M. et ft. pil.

    Sig.: One once or twice daily.

Stomach (irritability and acidity).

    ℞   Sod. chlor............................ ℨ ij.
         Sod. phos............................ ℨ iv.
         Sod. sulph........................... ℥ x.

    M. et sig.: Teaspoonful in seltzer twice daily an hour before eating.

General tonic in simple neurasthenia.

    ℞   Strych. sulph......................... gr. $\frac{1}{128}$.
         Ferri sulph........................... gr. $\frac{3}{4}$.
         Quin. sulph.......................... gr. j.
         M. et ft. elixir.................q. s. ad fl. ℥ j.

    Sig.: Teaspoonful after meals.

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(1)

THE END

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Professor of Clinical Medicine and Diseases of the Chest, College of Physicians
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